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OF THE OKLAHOMA STATE MEDICAL ASSOCIATION

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Editorials

A New Section in The Journal

The Editors of the *Journal* and a committee appointed by Doctor Everett, Dean of the School of Medicine and Director of the Medical Center, have evolved a plan to expand the usefulness of the *Journal* to its readers by devoting a section to the work going on at the Medical Center. It will be a kind of progress report to the physicians of the State without whose help progress would not be possible; but in addition it will include material designed to increase the readers basic knowledge of human disease and behavior particularly in those areas where important changes in concept are occurring.

The Medical Center includes the University of Oklahoma School of Medicine which is responsible for Medical education in Oklahoma and graduates nearly 100 young men and women each year; the University Hospital and the Children's Memorial Hospital which offer in-patient and out-patient service to patients who are the clinical material for teaching students and house officers; the Oklahoma Medical Research Foundation and its hospital which is concerned with both basic and clinical research; the Veterans Administration Hospital which is concerned with service to veterans, with research and with the training of house officers and students; the University of Oklahoma School of Nursing which offers to young women both a degree and a diploma program. Other facets of this soundly developing center which are perhaps less well known are: an extensive educational program for students working toward graduate degrees in the basic sciences; a program for the training of technologists of many kinds; a Speech and Hearing Clinic in a new building of its own; and many others.

It is difficult to comprehend the scope of the integrated whole of the Medical Center. Much of the work done is of such a basic nature that it must be reported in publications devoted to basic research. Many of the clinical studies are best reported in Journals whose readership is eminently qualified to criticize and to test. Consequently the members of the profession in Oklahoma who are asked to support the Medical Center in every way possible have little opportunity to know actually what is going on there.

This section is designed to correct this defect and to make it possible for a physician anywhere in Oklahoma to speak with knowledge to his friends about all parts of the Medical Center voluntarily or in answer to questions. The observation of a bacteriologist, Alexander Fleming, that a certain mold prevented the growth of streptococci on an agar plate required nearly twenty years to grow into a useful antimicrobial agent. With the help of the physicians and the people of Oklahoma who knows into what mighty oaks the little acros being planted at the Medical Center may grow.

Cancer Survival

There has been much controversy recently regarding various concepts of cancer therapy. This has raged most furiously over the mode of treatment for breast cancer.

Smith[1] summarizes the whole problem as follows: "The principle cancer therapy is the eradication of all tumor from the largest amount of tissue at the earliest possible moment. The accomplishment of the method of treatment is the amount of tissue cleared of tumor. The rate of survival does not reveal the accomplishment of the method of treatment. The rate of survival expresses the ratio of tumor cell distribution to the amount of tissue cleared of cancer. The survival rate varies principally with the unpredictable distribution of tumor cells.

When the difference in the accomplishment of two methods is slight, a comparison of survival rates cannot be expected to reveal that difference. Nevertheless, it is invariably true that superiority attends the method most nearly fulfilling the objectives of the principle of treatment. Therefore, compliance with the principle of treatment rather than comparison of survival rates should determine the method of therapy."

Another most interesting proposal regarding cancer survival has been suggested by Schrek.[2] He points out that life insurance statisticians have long used the average survival time for a given age to compute life expectancy. He shows that a modified form of average survival time—the median survival time (or its converse, the median death time)—can be used to evaluate cancer therapy results. Median survival time is the time at which 50 per cent of the patients in a given group remain alive. Median death time is similar to the laboratory measurement whereby results are expressed as terms of LD_{50}. Schrek suggests that the measurable virable be changed from dosage (as in LD_{50}) to time expressed as LT_{50}. Thus the lethal time for 50 per cent would be the the interval during which half of the patients in a given series die.

The use of LT_{50} would avoid the mistake of computing the average duration of life from the records of patients who have died from five to seven years after treatment, an error which is inherent in cancer statistics which are based upon survival rates rather than survival times.—J.M.

REFERENCES

1. Smith, J. Chandler: The Treatment of Cancer of the Breast, Surg., Gynec. and Obst., 104:36 (Jan.) 1957.

2. Schrek, R.: Fifty Per Cent Survival Time as a Measure of Prognosis in Cancer. Am. J. Clin. Path., 26:172 (Feb.) 1956.

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Scientific Articles

The Effects of Digoxin on CARDIAC OUTPUT *and* HEMODYNAMICS *in Experimental Supravalvular Mitral Stenosis**

WILLIAM E. JAQUES, M.D. and ALBERT L. HYMAN, M.D.

It is generally accepted that digitalis produces an increased cardiac output in congestive heart failure from mitral stenosis.[1] However, the occurrence of pure mitral stenosis in man is unusual if indeed it does exist. The opportunity to study the hemodynamics in these individuals is very limited.

In order to pursue this problem, a method was devised to produce heart failure from a pure supravalvular stenosis of the mitral valve in dogs.[2]

Material and Methods

Seventeen mongrel dogs were used in this experiment. They were caged separately and fed the routine diet. A supravalvular stenosis was produced as described previously.[2] The dogs were anesthetized with a 30 mg./kg. solution of Nembutal[R]**. The animals were studied during the control period with cardiac catheterization, femoral artery cannulation, oxygen consumption, and arteriovenous oxygen were determined. The cardiac output was determined by the Fick Principle. The rate of sodium and water excretion was determined in eight dogs.

After control studies were completed, 0.25 to 0.55 mg. of Digoxin[R]* was administered intravenously in all except four dogs. Two animals received 1.3 mg. of digoxin. The cardiac output, arteriovenous oxygen differences and pressures were recorded following the digoxin.

Two animals were phlebotomized of 150 and 270 ml. respectively.

Results

The acute response to intravenous digitalis was observed in 15 dogs (Table I and Chart I). In 12 animals, there was a significant fall in cardiac output. This was associated with a fall in pulmonary artery pressure in all but one animal. In this animal, the mean pulmonary artery pressure was 8 mm. Hg. before digitalization and 9 mm. Hg. after digitalization. In the 10 instances in which pulmonary "wedge pressures" were determined, it was only appreciably elevated in two dogs. In both these cases, the "wedge pressure" fell following digitalis. The total pulmonary vascular resistance, as measured at the pulmonary artery level, was significantly increased in six animals. The systemic arterial resistance was increased in all but one animal. There was no significant alteration in heart rate.

AUTHOR'S NOTE

William E. Jaques, M.D., graduated from McGill University Faculty of Medicine in 1942. His practice is limited to his specialty, Pathology. He has been certified by the American Board of Pathology and at present is Chairman and Professor of Pathology at the University of Oklahoma School of Medicine.

Doctor Jaques is a member of the American Society Experimental Pathology, the American Association of Pathology and Bacteriologists, the International Academy of Pathology and the American Society of Clinical Pathology.

Albert L. Hyman, M.D., graduated from Louisiana State University School of Medicine in 1945. Doctor Hyman has been certified by the American Board of Internal Medicine and his specialty is cardiology.

At the present time, Doctor Hyman is Assistant Professor of Medicine at Tulane University, New Orleans. He is a member of the American Federation of Clinical Research, a Fellow of the American College of Physicians, a Fellow of the College of Chest Physicians, and a Fellow of the American College of Cardiology.

CHART 1: THE EFFECT OF DIGOXIN ON THE

DOG	WEIGHT (Kg)	DOSAGE OF DIGITALIS (mgms)	CARDIAC OUTPUT IN L/MIN BEFORE DIGITALIZATION	CARDIAC OUTPUT IN L/MIN AFTER DIGITALIZATION	CHANGE	MEAN PULMONARY ARTERIAL PRESSURE IN MM/Hg BEFORE DIGITALIZATION	MEAN PULMONARY ARTERIAL PRESSURE IN MM/Hg AFTER DIGITALIZATION	PULMONARY WEDGE PRESSURE IN MM/Hg BEFORE DIGITALIZATION	PULMONARY WEDGE PRESSURE IN MM/Hg AFTER DIGITALIZATION	RIGHT ATRIAL PRESSURE IN MM/Hg BEFORE DIGITALIZATION	RIGHT ATRIAL PRESSURE IN MM/Hg AFTER DIGITALIZATION
98	9.0	0.80	5.00	3.00	Decrease	30.0	22.0	15.0	11.0	3.0	3.0
18	7.0	0.44	0.54	0.33	Decrease	8.0	9.0	5.0	4.0	E.D. 0	—
18	7.0	1.30	2.60	0.95 1.60	Decrease	23.0	19.0	8.0	—	E.D. 14.0 8.0	—
43	7.4	0.44	3.40	2.00	Decrease	35.0	28.5	14.0	13.0	E.D. 15.0	E.D. 3.7
42	8.3	0.25	3.10	1.50	Decrease	30.0	12.0	10.0	—	E.D. 15.5	—
96	8.0	0.50	1.40	0.64	Decrease	21.0	18.0	10.0	8.0	E.D. 8.0	E.D. 5.0
86	9.2	0.45	3.90	2.80	Decrease	23.0	19.0	4.0	—	E.D. 10.0	—
83	8.1	0.50	2.40	2.30	No change	18.0	17.0	7.5	5.0	E.D. 3.0	E.D. 5.0
77	7.9	1.30	2.60	1.20	Decrease	31.0	22.0	7.5	—	E.D. 4.0	E.D. 0
72	11.4	0.25	5.10	2.00	Decrease	30.0	25.0	15.0	—	E.D. 12.5	E.D. 10.0
62	12.8	0.80	2.70	0.69	Decrease	24.0	19.0	9.0	9.0	E.D. 9.0	E.D. 2.0
15	9.1	0.50	2.10	3.40	Increase	19.0	16.5	8.0	6.0	E.D. 4.0	E.D. 4.0
38	8.5	0.60	4.00	2.10	Decrease	37.0	24.0	23.0	10.0	E.D. 6.0	E.D. 0
70	10.3	0.50	3.20	1.90	Decrease	22.0	10.0	1.0	1.0	5.0	1.0
106	8.3	0.50	2.50	2.70	No change	35.0	27.0	25.0	18.0	E.D. 3.0	E.D. 3.0
		Amount of blood withdrawn			PHLEBOTOMIZED DOGS						
77	7.9	150.0cc.	2.00	1.28	Decrease	19.0	14.0	9.0	6.0	3.0	3.0
62	12.8	270.0cc.	3.50	2.70	Decrease	22.5	18.0	7.5	7.5	2.5	0

LEGEND
E.D.: Right ventricular end-diastolic pressure
—: Not recorded.

Graphic Representation of Cardiac Output

In only one dog was there an increase of 60 per cent in cardiac output after digoxin. There was a slight decrease in pulmonary artery pressure associated with a slight fall in pulmonary artery resistance and a slight fall in systemic arterial resistance. The pulmonary "wedge pressure" was not altered.

Two dogs showed no change in cardiac output. One of these animals displayed a fall in pulmonary artery pressure and "wedge pressure." In this dog the pulmonary artery resistance likewise fell, while the systemic arterial resistance rose slightly.

The hemoglobin varied from 9.2 to 14.9 gm.%, except in one animal when it was 7.8 gm.%.

The two dogs subjected to phlebotomy showed a decrease in cardiac output and a fall in pulmonary artery pressure. There was no appreciable change in pulmonary "wedge pressure" which had been normal. The pulmonary vascular resistances showed no appreciable change while there was definite increase in systemic arterial resistances. There was a slight fall in hemoglobin.

The sodium and water excretions were determined in eight dogs following digitalization (Table II). In four animals, there was a pronounced naturesis and diuresis. Four animals showed no change and one displayed a slight decrease after digitalis. These changes could not be correlated with a change in cardiac output, or right ventricular end-diastolic pressure.

Discussion

These experiments were designed to test the effect of digitalis in a pure supravalvular

HEMODYNAMICS IN EXPERIMENTAL MITRAL STENOSIS

PULMONARY ARTERIAL RESISTANCE IN DYNE/CM-5		PULMONARY ARTERIOLAR RESISTANCE IN DYNE/CM-5		LEFT SIDED RESISTANCE IN DYNE/CM-5		SYSTEMIC ARTERIAL RESISTANCE IN DYNE/CM-5		HEART RATE		Hb (gms%)
BEFORE DIGITALIZATION	AFTER DIGITALIZATION	BEFORE DIGITALIZATION	AFTER DIGITALIZATION	BEFORE DIGITALIZATION	AFTER DIGITALIZATION	BEFORE DIGITALIZATION	AFTER DIGITALIZATION	BEFORE DIGITALIZATION	AFTER DIGITALIZATION	
490.0	710.0	260.0	350.0	230.0	360.0	1800.0	5100.0	200.0	200.0	12.0
1280.0	2180.0	480.0	1210.0	800.0	970.0	1850.0	2900.0 2580.0	160.0	130.0	11.5
770.0	1200.0	510.0	—	260.0	—	4500.0	7700.0	170.0	180.0	10.5
830.0	1140.0	500.0	980.0	330.0	160.0	2690.0	4350.0	165.0	150.0	9.4
775.0	640.0	520.0	—	255.0	—	3500.0	4140.0	175.0	175.0	11.4
1200.0	2250.0	630.0	1250.0	570.0	1000.0	5800.0	19000.0	180.0	165.0	12.0
480.0	550.0	390.0	430.0	90.0	120.0	2700.0	2700.0	150.0	150.0	9.4
670.0	600.0	430.0	420.0	240.0	180.0	4950.0	4700.0	150.0	155.0	7.8
1270.0	1460.0	970.0	970.0	300.0	490.0	6400.0	9000.0	170.0	150.0	9.2
475.0	1000.0	235.0	—	240.0	—	2300.0	6600.0	165.0	175.0	13.5
460.0	2340.0	250.0	1280.0	190.0	760.0	4350.0	20000.0	160.0	160.0	11.5
725.0	390.0	305.0	248.0	420.0	142.0	5210.0	3200.0	165.0	150.0	11.8
740.0	920.0	112.0	535.0	628.0	385.0	4940.0	6900.0	180.0	200.00	12.1
501.0	416.0	490.0	375.0	11.0	41.0	3400.0	9680.0	200.0	200.0	14.9
1150.0	740.0	320.0	160.0	830.0	160.0	3650.0	4820.0	180.0	175.0	15.0
760.0	880.0	400.0	500.0	260.0	330.0	4330.0	5500.0	150.0	150.0	12.5
530.0	560.0	390.0	250.0	140.0	130.	3650.0	4500.0	150.0	150.0	12.5

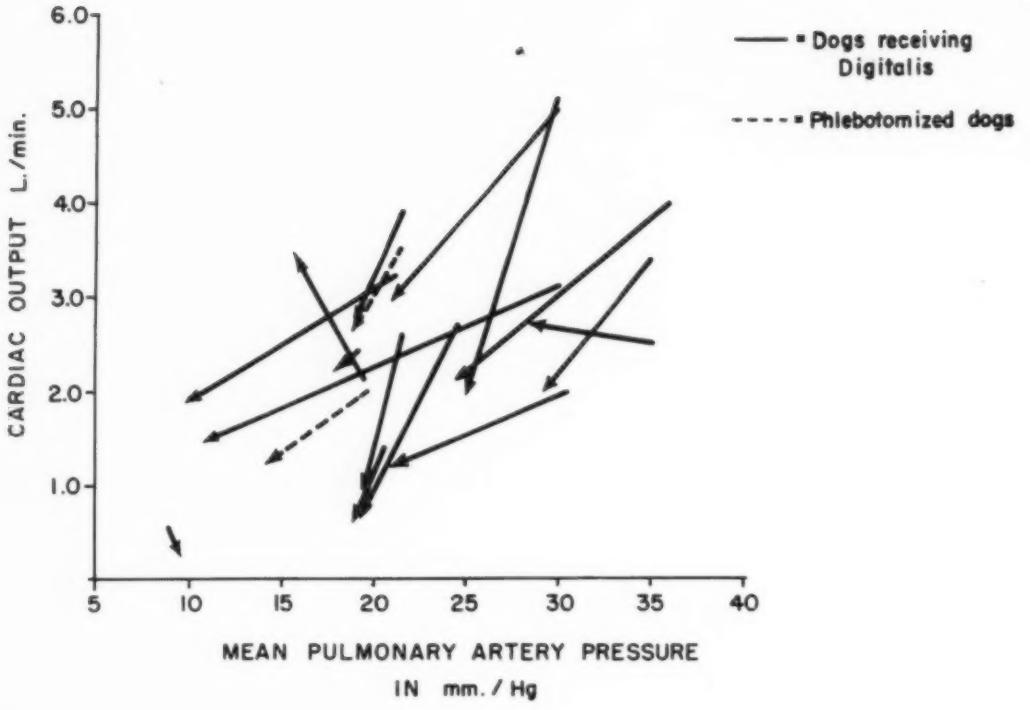
Output and Mean Pulmonary Artery Pressure

mitral stenosis without any element of mitral regurgitation of myocardial damage. The only clinical and anatomic evidence of myocardial damage was noted where the suture encircled the left atrium.

The results indicate that within five to ten minutes after the administration of digitalis, in most instances, the pulmonary artery pressure falls and during the ensuing 30 to 60 minutes there is a fall in cardiac output.

It seems probable that in these experiments the digitalis produces a systemic or splanchnic venous vasodilatation.[3, 4] A fall in pulmonary artery pressure followed. With a decrease in pressure, the flow through a fixed stenotic valve fell. Indeed, by reducing the venous return to the heart with rapid phlebotomy, similar observations were obtained.

Numerous observations on the effect of digitalis on patients with rheumatic mitral stenosis and heart failure have indicated a rise in cardiac output with a fall in right heart pressure.[1] The dissimilarity between these results and our findings may perhaps be related to actual myocardial damage by rheumatic fever or to an unrecognized element of mitral regurgitation or both. The purer the mitral stenosis and the less the myocardial damage by rheumatic heart disease, the less likely it is that the cardiac output will be increased by digitalis. Ferrer, and colleagues[5] found no changes after digitalization of patients suffering from mitral stenosis with a mechanical block only. Their data indicate that digitalis is only helpful if myocardial damage is present.



The observations of sodium and water excretion fail to reveal any apparent correlation between the latter and cardiac output. There were frequent instances of a fall in cardiac output with a diuresis and naturesis. In all these animals, the right atrial or right ventricular end-diastolic pressure was normal before digitalization and was not appreciably altered by digitalization. The finding of normal atrial pressure in dogs with heart failure from experimental mitral stenosis has been previously reported.[6] There was no apparent correlation of the latter pressures with the excretion of sodium and water in the urine. Previous observations from this laboratory indicated that digitalis exerts a direct effect on the kidney.[7] It is possible that this mechanism can explain the naturesis and diuresis frequently observed.

Continued on Page 159

Dog	Wt. in Kg.	Dosage of Digitalis	Cardiac output before Digitalis in L/min.	Cardiac output after Digitalis in L/min.	Highest total No excretion before Digitalis in meq./min.	Highest total No excretion after Digitalis in meq./min.	Greatest urine volume before Digitalis in ml./min.	Greatest urine volume after Digitalis in ml./min.
98	9.0	0.80	5.00	3.00	1.00	2.60	0.10	0.13
15	9.1	0.50	2.10	3.40	1.17	1.23	0.42	0.51
38	8.5	0.60	4.00	2.10	0.23	1.30	0.14	0.70
70	10.3	0.50	3.20	1.90	0.29	0.38	0.19	0.19
18	7.0	1.30	2.60	0.95	0.50	0.52	0.25	0.50
77	7.9	1.30	2.00	1.20	0.54	0.83	0.12	0.15
83	8.1	0.50	2.40	2.30	4.70	0.40	0.27	0.12
72	11.4	0.25	5.10	2.00	1.30	1.20	0.07	0.10

Table II
Representation of Relationship Between Cardiac Output and Water And Sodium Excretion

The Control of a Post-Natal Epidemic of BREAST ABSCESSES *in a* GENERAL HOSPITAL

ROBERT W. DEAN, M.D.

THE AUTHOR

Robert W. Dean, M.D., graduated from the University of Oklahoma School of Medicine in 1947. He is now a resident in Obstetrics and Gynecology at St. John's Hospital, Tulsa, Oklahoma.

This paper was presented at the Third Annual Meeting of the Oklahoma Association of House Staff Physicians, May 31, 1957 in Oklahoma City.

In the fall of 1956 it became apparent that an epidemic of breast infections existed at St. John's Hospital when one obstetrician noticed that abscesses developed in six of his post partum patients in a short period of time. Upon questioning several obstetricians, he found that they also were encountering this problem. Largely through his efforts, measures were taken which have markedly reduced the incidence of this post partum complication at our hospital. The number of breast infections is shown in Figure I, with his cases shown above the total number for the hospital.

A committee was organized to study this problem and the problem of hospital infections in general. The members of the committee were three obstetricians, the obstetrical resident, the obstetrical supervisor, two pediatricians, two surgeons, an internist, a pathologist and an otorhinolaryngologist. This group gathered data on all hospital infections; reviewed relevant journal articles and studied current hospital practices. It made many valuable suggestions for the correction of breaches in technique.

Articles which came in contact with either the mothers' breasts or the infants during the period of hospitalization were cultured. A representative list included: infant oxygen masks, sterilizing solutions, cord ties, breast pads, and nursery stethoscopes. Of all formites cultured, only the stethoscopes were found to harbor pathogenic organisms. These stethoscopes were subjected to zephiran cleansing after being used each time, and on subsequent cultures were found to be negative.

Throat, nasopharyngeal and hand cultures were taken of all obstetricians, pediatricians, nurses, student nurses, aides and maids. The incidence of positive cultures is shown in Figure II. The treatment of persons whose cultures were positive was carried out under direction of the otorhinolaryngologist on the committee, and each carrier was followed until his culture remained negative. It was necessary to discharge one aide whose work habits did not improve as per suggestion and who did not accept treatment.

A questionnaire was prepared and mailed to each obstetrician in order to include all known cases of mastitis and breast abscesses. The response to the questionnaire was 100 per cent. Although cases of mastitis and breast abscess were listed separately on the questionnaire as shown in Figure III, no distinction was made in tabulating them in Figure I because basically they represented differing degrees of the same entity. The delivery room technique was subjected to careful scrutiny. Several careless practices had developed in our delivery room. This was due to the previous absence of obstetrical infections and to the generally complacent attitude towards infection in this antibiotic era. Obstetricians wearing street clothes would occasionally enter the delivery room to observe an intern's delivery technique. Anesthesiologists and circulating nurses were not wearing masks when in delivery rooms. Babies were being cleansed with baby oil only.

The corrective measures are obvious. No one was allowed in the delivery room in street clothes or without masks. Phisohex was substituted for baby oil in cleansing the newborns. Cooperation was excellent and few enforcement measures were necessary.

Our newborn nursery consists of two large rooms with a workroom between them. Each room contains forty individual crib units

	Nasopharyngeal Cultures				Hand Cultures
	Doctors	Students & RN's	Aides & Maids	Total	All Personnel
Cultures (#)	46	49	68	163	84
Positives (%)	22	18	16	18	1

Nasopharyngeal and Hand Cultures Taken in December

Figure II

separated by clear plastic dividers. Although each crib unit has its own linen stack, we found that nurses aides were using the linen stacks from crib units of babies who had gone home before they had been resterilized. About three years ago the pediatricians complained that infants were being burned by the ultraviolet germicidal lights so these were turned off. We turned these back on. The nursery personnel was instructed to wear masks and to change them several times during an eight hour shift. Periodic meetings were held with all nursery personnel to stress the value of aseptic technique with emphasis on frequent hand washing. Throat cultures were done on all breast fed babies on the fourth day so that the pediatrician and obstetrician could be appraised of positive cultures and institute preventive measures early. Figure IV shows the results of these cultures. The nasopharynx of every baby in the nursery was cultured on February 20th; there were no positive cultures.

It was recommended that breast feeding be stopped. This was an important factor in stopping the epidemic because 95 per cent of the infections occurred in nursing mothers. Preventive measures were not generally adopted until about the last week in December when the epidemic nature of the problem was apparent to all. However, the obstetrician who had the six cases near the onset of the epidemic, and who previously estimated that 50 per cent of his mothers nursed, stopped virtually all of his patients from nursing about the third week in November. Comparing this man's series of cases with the general epidemic closely approximates a controlled study since he began discouraging nursing a month prior to the general institution of all described preventive measures. This comparison is illustrated in Figure I.

	Total Cultures	Positive Cultures
Dec. 11-Dec. 17	3	1
Dec. 18-Jan. 3	8	1
Jan. 4-Jan. 18	21	1
Jan. 19-Feb. 3	24	3
Feb. 4-Feb. 20	22	0
*Feb. 20	60	0

Nasopharyngeal Cultures On All Nursing Babies Taken on Fourth Hospital Day

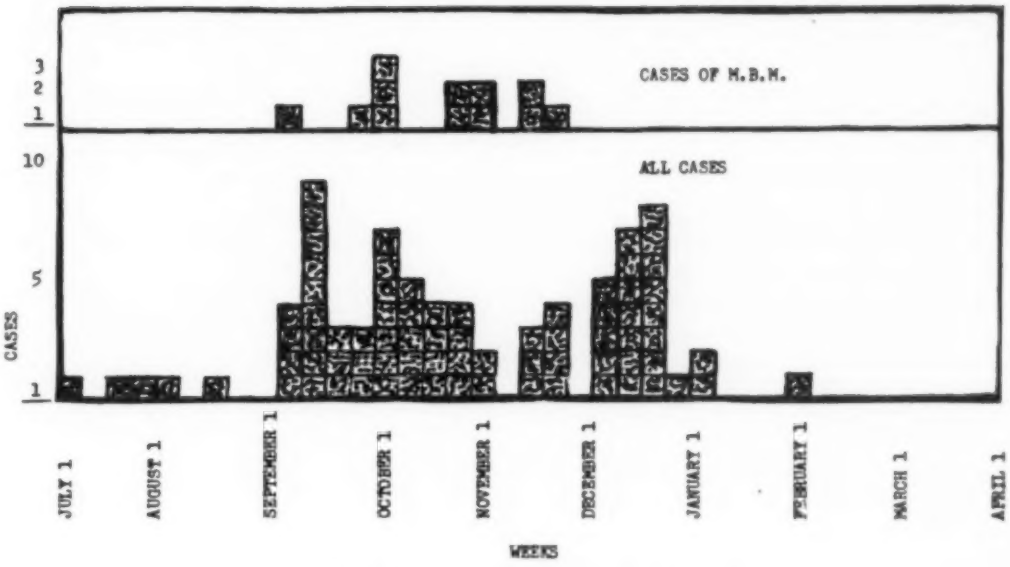
*All Babies in Nursery Cultured On This Date

Figure IV

Another measure thought to be of value was early release from the hospital of nursing mothers. Since this was thought to be a hospital epidemic, it was recommended that all mothers who insited on nursing be encouraged to leave on the third hospital day. All charity cases were discharged on the second or third post partum day, as compared to an average stay of five to seven days for private patients. It is of interest, therefore, to compare the incidence of these two groups, which is 1.44 per cent for charity patients and 5.31 per cent for private patients during the period from September 1st to December 31st, 1956.

PATIENT	DATE OF DELIVERY	DATE OF ONSET OF BREAST SYMPTOMS	MASTITIS ONLY		ABSCESS NOT DRAINED		ABSCESS DRAINED		ANTIBIOTIC USED	RESULTS OF CULTURES AND SENSITIVITIES	PATIENT NURSED		DATE OF I & D	PEDIATRICIAN	OBSTETRICIAN
			RT	LT	RT	LT	RT	LT			YES	NO			

Figure III
Questionnaire Sent To Obstetricians And General Practitioners



BREAST INFECTIONS FROM JULY 1 TO APRIL 1, INCLUSIVE
FIGURE I

The epidemic and its abrupt cessation are graphically illustrated in Figure I. The incidence of breast infections between September 1st and December 31st, 1956 is 4.93 per cent. This compares to an incidence of 0.62 per cent between July 1st and August 31st, 1956 and to an incidence of 0.1 per cent between January 1st and March 31st, 1957. Because only about 50 per cent of our staff actively discouraged nursing and encouraged early discharge of nursing mothers, we feel justified in attributing the control of the epidemic to improvement in delivery room and nursery asepsis, and to the vigorous treatment of carriers. We are gratified that these measures proved effective, and that widespread prophylactic use of antibiotics was not necessary. Some hospitals have given prophylactic antibiotics to all newborn infants while they were in the hospital. We feel that our approach is more rational.

Our experience brings into focus several important points. First, it shows that a resistant organism can emerge and cause an epidemic. Although only fourteen culture and sensitivity studies were done on drained absecesses, it is significant that all of these showed a penicillin resistant staphyloccus aureus. This again emphasizes the value of adequately treating every infection in order to develop fewer resistant organisms and shows the increasing necessity for culture and sensitivity studies in this day of multiple antibiotics and their promiscuous use.

The next thing that came to our attention was the number of breaches in aseptic technique. For this reason, we strongly recommend that every hospital organize an infection committee. Furthermore, there was a surprising incidence of carriers among our hospital personnel. We advise nasopharyngeal cultures, periodically, to control this problem.

In the event that any hospital should have an outbreak of breast infections similar to ours, we suggest that breast feeding be stopped immediately in so far as possible. Mothers who insist on nursing should be discharged on the third post partum day. An additional precaution thought to be of value is the use of nipple shields by nursing mothers. The prophylactic use of antibiotics is a debatable issue and we are not able to add any information on this point, however, they were not generally used for either the mothers or the infants in our study.

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STOP RHEUMATIC FEVER

WILLIAM W. SCHOTTSTAEDT, M.D.

THE AUTHOR

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This is the seventh in a series of articles prepared for *The Journal* under the auspices of the Oklahoma State Heart Association and its Committee on Professional Education, emphasizing the theme of the Association for the current year.

The program to STOP RHEUMATIC FEVER has focused attention upon prompt and adequate treatment of upper respiratory infections due to beta-hemolytic streptococci and prophylactic treatment of patients who have once had rheumatic fever. Such therapeutic and preventive measures are useful because the epidemiology of rheumatic fever is the epidemiology of the beta-hemolytic streptococcus. And of the various streptococci, Group A have been found to be the most frequent cause of pharyngeal infection. Yet not all persons with Group A streptococci develop symptoms. Many do develop a febrile response which causes them to seek medical care; some 20 per cent, however, develop a sore throat with no constitutional symptoms. And an unknown but appreciable number develop no symptoms whatever. Of those who have a febrile response, only about three per cent then go on to develop acute rheumatic fever. These are fascinating facts and figures. Can one learn by a study of the 97 per cent who do not develop rheumatic fever what accounts for their resistance to the disease? Would new measures to STOP RHEUMATIC FEVER be found if one could but learn why some individuals develop no signs or symptoms of illness despite the presence of Group A streptococci in their nose and throat? Clearly, these are questions which deserve attention.

At the turn of the century, many studies were done on environmental factors in rheumatic fever. These were summarized by John R. Paul[1] in 1943. The geographic distribution of rheumatic fever seemed to indicate that cold, wet climates were a factor in increasing susceptibility, though unusually cold winters were not associated with an increased incidence of the disease. A seasonal peak was observed: March and April in the New England states, January and February in Oklahoma, and November in England. But studies failed to show any correlation with humidity or percipitation and the effects observed could have been related to crowded living. Attempts to establish this possibility, however, by analysis of incidence of rheumatic fever in relation to population density showed no significent correlation except for an increased incidence in the urban population which was subject to many interpretations. The incidence was higher in low-income groups, but not strikingly so. The studies of Quinn[2] have shown that crowding is associated with an increased incidence of rheumatic fever, whether in an urban or a rural setting.

Another environmental factor which has been studied is the inanimate reservoir for streptococci: dust, clothing, and the like. Coburn and Young[3] reported survival of some strains of streptococci for as long as eight months in ordinary dust. However, they felt that human reservoirs were more important in the spread of streptococci. This was borne out by the studies of Wannamaker[4] who found no higher incidence of streptococcal infection among servicemen using blankets contaminated with large numbers of streptococci than among those using clean blankets.

But studies of streptococci and of the environment have not demonstrated any factor or combination of them capable of producing the diverse effects known to follow streptococcal infection. Man has some degree of

resistance which in some undefined way determines whether a transient or chronic carrier state, an inapparent infection, or clinical illness develops after exposure to beta-hemolytic streptococci and whether or not rheumatic fever follows. The interaction which occurs between the streptococcus and its host, man, involves a multiplicity of antigenic substances, bacterial toxins, and substances related to virulence on the one hand and an equally formidable array of metabolites, antibodies, and host factors toxic to the streptococcus on the other. It seems clear from animal experimentation that, in addition to these factors which affect the immediate outcome of host-parasite relationship, certain factors in the host are essential to the production of rheumatic-like lesions.

Many antibody responses have been defined and studied in man, most prominent among these being the antistreptolysin-O titer. Stetson[5] has reported that, despite great individual variation, subjects who develop rheumatic fever ordinarily show a greater antibody response to streptococcal infection than do other subjects. Bernheimer[6] has suggested a relation to more frequent streptococcal infections among the rheumatic subjects but Cantanzaro, Stetson and colleagues[7] demonstrated that rheumatic fever could be suppressed by controlling streptococcal infection without suppressing the ASO response. The immune response was therefore not a primary event in the development of rheumatic fever.

The carrier state has also been investigated. Rammelkamp[8] has described the ordinary course of an untreated infection. Group A streptococci gradually disappear and after three or four weeks can no longer be recovered from cultures of the nasal membranes. Occasional colonies persist in the pharynx, however, and the majority of untreated subjects continue to harbor organisms for at least four months. Wannamaker[4] showed that nasal carriers were more important in dissemination of streptococcal infection than throat carriers because the streptococci in the latter tend to lose the ability to form M-protein, which is essential to infectivity. Nevertheless long survival in the oropharynx does seem to be important for other reasons than dissemination of infection. In the opinion of Rammelkamp rheumatic fever does not occur unless streptococci persist in the oropharynx.

It has been known for many years that rheumatic fever was more apt to occur in families where it had already been present. Parents who had had the disease were more apt to have children who developed it than parents who had not, but identification of true hereditary factors has been difficult. George Griffiths[9] has reported a very interesting study of this facet of rheumatic fever. Though children of parents who have had rheumatic fever are more apt to develop the disease, this is only true if they maintain contact with their family. The incidence of rheumatic fever was not higher among children of rheumatic parents if the individual had left his family. The difference in incidence was therefore not hereditary but lay somehow in the household or family.

Three medical students of the Oklahoma University School of Medicine have recently carried out a pilot study[10] which seems to provide another possible link in this long chain of events which ultimately leads to the development of rheumatic fever. Grouping and typing of streptococci were not done but, with this limitation, the data indicated that even intimate exposure did not result in the appearance of streptococci in throat cultures of all individuals exposed; but a correlation appeared to be present between stresses affecting the security of the family and the appearance of positive cultures. Since no clinical infections occurred during this study the data are of limited significance. They suggest however that another set of factors may also be important in this problem, especially in view of Griffiths finding that the familial incidence of rheumatic fever was domiciliary and not hereditary.

The numerous factors which are important in determining the interrelationship between man and the streptococcus deserve careful attention in the hope of adding new avenues by which we may STOP RHEUMATIC FEVER.

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Splenectomy for Congenital HEMOLYTIC ANEMIA

ROBERT JABOUR, M.D.

THE AUTHOR

Robert Jabour, M.D., graduated from the University of Tennessee School of Medicine in 1954. Doctor Jabour is now a resident in General Surgery at Hillcrest Hospital in Tulsa.

This paper was presented at the Third Annual Meeting of the Oklahoma Association of House Staff Physicians, May 31, 1957, in Oklahoma City.

Quoting from Dameskek, "The spleen remains an organ of mystery. It seems to be a part of the endrocrine system; of this real proof is lacking. It may be removed from the body with impunity, but it has many subtle functions connected chiefly with the bone marrow. Ever so often, particularly when enlarged, it seems to bring about serious blood dyscrasis, and its removal may then become essential."

Splenectomy is almost specific in the treatment of Congenital Hemolytic Anemia, which is characterized by *intermittent jaundice*, an *enlarged spleen*, and *periods of weakness and fatigue*. Acute attacks may occur, manifest by *upper abdomnial pain, nausea, vomiting, fever, increase of anemia and jaundice*, simulating an acute surgical abdomen.

Sperocytosis, inherited as a Mendelian dominant trait, is believed responsible for the main features of the clinical disease. These sperical red cells are more fragile than the biconcave discs, and the clinical symptoms appear to be due to the rapid destruction of the spherocytes by the overactive spleen. In an acute hemoclastic crisis, there is a rapid fall in red cells within 24-48 hours. These crises may be precipitated by infections, operations, and other stress situations. Transfusions, may at times, stimulate the overactive spleen to increase its phagocytic function.

Onset of the clinical disease usually occurs during childhood or early adult life. It has been said that the younger the patient at the time of the first crisis, the more severe the disturbance tends to become. Gallstones are a frequent complication, occurring in approximately 60 per cent of cases.

In addition to the clinical picture, corroborative laboratory findings include *spherocytosis, a variable degree of anemia, reticulocytosis, an elevated serum bilirubin, increased fragility of the red cells* and a *negative Coombs test*. Splenectomy should be done in those patients in whom the diagnosis can be established. Following splenectomy, the *jaundice subsides, the anemia disappears*, and the *reticulocytes return to normal*. The spherocytosis (and often the increased fragility of the red blood cells) persists.

Case Reports

CASE I: A white male, aged 15, admitted to the hospital with complaints of pain in the mid-epigastrium, nausea and vomiting. The onset of pain was said to have occurred approximately 48 hours prior to admission, and following the ingestion of hamburgers with onions. The pain was said to be colicky in nature and gradually increasing in severity. The patient stated he had a somewhat similar episode three months previously, which had been of only 12 hours duration, and which was relieved by vomiting and bicarbonates. The day after the onset of the present illness, the patient was noted to be jaundiced. The patient stated that he had a swelling in the left upper part of the abdomen which was not tender. Clay colored stools and dark urine were noted the night prior to admission.

An interesting family history revealed:

1) The father, aged 46, had a history of intermittent right upper abdominal pain, jaundice, weakness, fatigue, and inability to hold jobs because of illness.

2) An uncle, aged 45, had recently had a splenectomy, and also had cholelithiasis for which plans for an operation were being made.

3) An aunt, aged 47, who had a cholecystectomy in 1947, has had recurrent bouts

with jaundice and abdominal pain.

4) A cousin, aged 12, had been hospitalized for an anemic condition three years previously. He had failed to respond to conservative therapy, including transfusions, and a splenectomy had been performed with excellent results.

Physical Examination: Revealed a well nourished, well developed but acutely ill boy. His temperature was 100² F. Patient was in moderate pain at the time. The patient was deeply jaundiced. The abdomen was scaphoid. The liver was not enlarged, but there was some tenderness to fist percussion over the right costal margin. The spleen was enlarged four fingerbreadths below the left costal margin, was firm and non-tender. The rest of the abdominal examination was negative. A putty-colored stool was noted on rectal examination.

CASE I:

RBC	—	3,290,000	cells cu/mm
WBC	—	11,800	" "
Hgb.	—	11.7	gms.
Reticulo.	—	10.3	
Spherocytes on the Direct Smear			
Increased RBC fragility		(0.69%	— 0.39%)
Coombs	—	Negative	
Ceph. Flocc.	—	3+	
Van den Bergh			
Direct	—	9.7	mgm%
Indirect	—	14.7	"

Alkaline Phosphatase	—	11.05 units
Prothrombin Time		13.9 sec. —77%
Fecal Urobilinogen		340 Ehrlich Units (75-300)
Urine Urobilinogen		5.75 Ehrlich Units (0-1.25)
Albumin		4.83 gms. %
Globulin		2.32 gms. %
Protein		17.4 meq/l.

Figure 1

The patient was put on a high protein, high caloric, low fat diet, and treated conservatively for a period of 10 days, during which time there was improvement in clinical symptoms and subsidence of jaundice. Repeated X-ray examination, including flat plate, cholecystogram, and cholegraffin study failed to reveal the presence of biliary calculi.

Following this a splenectomy was done. The spleen was about five times the normal size and there were two accessory spleens. His course was uneventful. Figure II shows the change that had occurred by the sixth day.

Slide II

RBC	—	4,480,000	compared to		3,290,000
WBC	—	7,100	"	"	11,800
Hgb.	—	14.7	"	"	11.7
Platelets	—	650,000	"	"	365,000
Reticulo.	—	0.4%	"	"	10.3%
Van den Berg					
Direct	—	1.81	"	" 9.7	mgm%
Indirect	—	3.00	"	" 14.7	mgm%

The patient was discharged on the tenth day after operation. Three months later the patient was readmitted with right upper quadrant pain, colicky in nature, nausea, vomiting, clay colored stools and dark urine. Cholecystectomy was performed with exploration of the common duct. The gallbladder contained a few small bilirubinate stones. The patient was discharged on the twelfth post-operative day.

CASE II: The aunt of the patient described above was admitted in May 1956, two days after his discharge, following the splenectomy. She was 47 years old and was admitted with a history of malaise, muscular aches and pains and anorexia of four days duration. Six hours prior to admission she had a fairly sudden onset of severe pain in her right chest, accompanied by dyspnea, but no cyanosis. The patient also stated that she had had a chill just prior to the onset of pain in the chest. She denied any previous episodes of this nature.

Past History: There was a known family history of congenital hemolytic icterus. She had a hysterectomy in 1931 and a cholecystectomy in 1947. There had been recurrent bouts of jaundice, and abdominal pain without clay colored stools or dark urine since the chloecystectomy.

Physical Exam: Temp. 105, Pulse 120, Resp. 36, BP 140/55. The patient was slightly obese and appeared accutely ill. Her skin appeared anemic, with a suggestion of icterus. A friction rub was noted in the right lung field with rales noted both anteriorly and posteriorly. There was pain on deep respiration. The liver was not palpable. There was tenderness to palpation in the mid-epigastrium. The spleen was greatly enlarged, filling the entire left upper quadrant, and approximately seven fingerbreadths below the left costal margin, and was firm and non-tender.

CASE II

RBC	—	2,670,000
WBC	—	16,700
Hb.	—	8.7
Hct.	—	21. %
Platelets	—	320,000
Reticulo.	—	2.1%
Spherocytes on the Direct Smear		
Increased RBC fragility		
Coombs	—	Negative
Van den Berg		
Direct	—	0.60 mgm %
Indirect	—	4.40 mgm %

Figure III

The patient was treated for her pneumonic condition with good response. In the pre-operative period both washed red cells and whole blood transfusions were utilized. At operation a spleen 8-10x normal size and one accessory spleen were found. Examination of the common and hepatic ducts was not remarkable. Her post-operative course was very satisfactory.

CASE II—10 days after operation

RBC	—	4,240,000	compared	to	2,670,000
WBC	—	10,100	"	"	16,700
Hgb.	—	13.4	"	"	8.7
Hct.	—	42%	"	"	21%
Platelets	—	632,000	"	"	320,000

Figure IV

CASE III: Father of the first patient (Case I) was 46 years old. He gave a history of recurrent episodes of abdominal pain, jaundice, weakness, fatigue, and inability to hold jobs. His first attack dated back to adolescence. There was a history of food intolerance. His abdominal pain was said to occur in the mid-epigastrium, or in the right upper quadrant and would last approximately a week and then subside. The patient then noted belching, bloating or fullness for another two-three weeks. The patient's mother died with "yellow jaundice, dropsy and liver disease at age 39." He was a welder by profession, but due to his chronic debilitating condition, the patient and his family had a significant socio-economic problem.

Physical Examination: Revealed a fairly well developed white male, who appeared somewhat pale and apathetic. There was a slight icteric tint to the sclerae. The liver was palpable two finger breadths below the right costal margin, was firm and non-tender. The spleen descended two-three finger breadths below the left costal margin and was also firm and non-tender. No other significant physical finding.

CASE III

		Post.Op Lab Work
RBC	— 4,180,000	Ceph. Flocc. - 3+
Hgb.	— 12.2	A/G Ration
WBC	— 12,600	Albumin - 2.8
Reticulo.	— 1.2%	
Spherocytes on Direct Smear		Globulin - 4.0
Coombs — Negative		Total Prot. -16.4 meq/L
Total Bilirubin—1.7 mgm%		

Figure V

With but a slight elevation in bilirubin and reticulocyte count, and with a fairly normal hemogram, the optimal time for splenctomy seemed present. Marked venous congestion with evident portal hypertension was noted at operation. The liver appeared somewhat granular, firm and fibrosed, compatible with a diagnosis of biliary cirrhosis. The gallbladder was markedly enlarged and tense. No calculi were palpable but cholecystic disease was present. The spleen was three times normal size, and densely adherent to the surrounding structures. These fibrous and calcific adhesions, later encountered, gave rise to considerable venous oozing, and made delivery of the spleen difficult with resultant raw surfaces. The immediate post-operative course was satisfactory, except for a persistent drainage for a few days. The patient's general course was good, and he was discharged on the tenth post-operative day.

Discussion

Each of the three cases presents interesting facets of this disease. The first, a 15 year old boy whose condition was diagnosed shortly after the first clinical manifestations of the disease, had the complications of cholecystitis and cholelithiasis.

The second presents a middle aged white female who previously had had a cholecystectomy but had had a recurrence of abdominal complaints and jaundice and a hemolytic crisis had been precipitated by a pneumonic condition.

The third case showed the possible effects of longstanding untreated congenital hemolytic disease, with its attendant complications, and resultant liver disease and portal hypertension.

(1151 Utica Square, Tulsa, Oklahoma)

Difficulties in Differential Between
Chorio-Adenoma Destruens *and* Choriocarcinoma

WILLIAM E. HOOD, Jr., M.D.

Hydatidiform mole is uncommon. It occurs in only one of 2,000 to 2,500 pregnancies. Its malignant counterparts chorioadenoma destruens and choriocarcinoma occur much more rarely. When it is remembered that the normal trophoblasts display invasion of the uterine wall and blood vessels for their very existence in normal pregnancy, it is not surprising that malignant forms exist. It is surprising that they are not more prevalent. The diagnosis of these tumors present many difficulties and pitfalls. It behooves all practitioners of obstetrics to be aware of these conditions and their import to the young female.

THE AUTHOR

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This paper was presented at the Third Annual Meeting of the Oklahoma Association of House Staff Physicians, May 31, 1957 in Oklahoma City.

History

Mrs. R. E. C., an 18 year old white female secretary, prima gravida, was first seen in the outpatient department of the U. S. Naval Hospital in Memphis, Tennessee, in December 1955. The chief complaint was amenorrhea of two months duration. Menstrual history revealed the menarche to be at age 13 years with the onset of a 28 day cycle. Menstrual periods were five days duration with moderate cramping dysmenorrhea the second and third days. Her last normal menses occurred November 4 through 9, 1955. The usual subjective symptoms of pregnancy were present and the uterus was found to be slightly enlarged, suggesting early pregnancy. On February 13, 1956 the patient noticed the onset of vaginal spotting with an increased amount of clear vaginal discharge. On March 25 severe pelvic pain preceded a profuse vaginal hemorrhage, followed by the passage of a large mass of "grape-like" tissue. Pathological examination of the tissue revealed a mass of cystic structures with a considerable amount of fresh and clotted blood. The specimen weighed 919 grams and measured 15 x 9 x 5.5 cm. Microscopic diagnosis was hydatidiform mole. On admission to the hospital a pelvic examination revealed uterine enlargement compatible with three to four months gestation. There was a separate soft cystic mass thought to be ovarian filling of the pelvis. On March 27, 1956 due to continued hemorrhage a curettage was performed. The currettings revealed fragments of decidua, blood clot and trophoblasts. No evidence of malignancy was reported. It was thought that treatment should be expectant. On June 4, 1956, three months after curettage, she consulted her physician in Oklahoma City, because of continued vaginal spotting and intermittent moderate bleeding. Pelvic examination revealed no uterine enlargement or adnexal massing. Moderate adnexal tenderness was present bilaterally. A Papanicolaou smear taken at this time was Class II with sheets of atypical columnar cells, many showing variation of nuclear size. A Friedman test was negative. On June 9 in St. Anthony's hospital at curettage the uterus was sounded to 2¼ inches. A softened area was noted in the left cornu during curettment. Bleeding was minimal. Microscopic examination revealed proliferative endometrium with large areas of hyalinization and partial calcification with inflammation. Hemoglobin on this admission was 11.4 grams. She was next seen on August 12, 1956 with marked bleeding, palpitation, dizziness, dyspnea and cramping pelvic pain of two weeks duration. Hemoglobin was 8.6 grams. On August 13 at curettage the cervix was 2 cm dilated. A large black rubbery mass appearing to be endometrial tissue was seen clinging to the cervix. Its removal resulted in brisk hem-

orrhage. The uterus seemed slightly enlarged but could be sounded only to 2¼ inches. Microscopic diagnosis was endometrium with groups of trophoblastic cells without necrosis. (Chorio-adenoma destruens and choriocarcinoma could not be excluded.) A Friedman test was positive although she denied absolutely on several occasions any possibility of pregnancy. On August 23rd a roentgenogram of the chest was negative. A quantitative Aschem-Zondek test for gonadotropic hormone excretion done on 23 day old rats was positive in dilutions up to 1:200,000. On October 8, 1956 a pelvic examination was essentially negative except for 3° uterine retroversion. Hemoglobin was 11.4 grams. On October 23 a repeat Ascheim-Zondek test was positive in dilutions up to 1:000,000. At this time the patient left Oklahoma City to join her husband now in California. She returned to Oklahoma City on December 8, 1956 with a history of being asymptomatic but amenorrheic. She did report a very light brown vaginal discharge on one or two occasions. On about December 1 she noted a severe generalized headache unrelieved by medication. During the next eight days three or four similar episodes occurred. These headaches were not localized. There were no additional complaints. On December 10 at approximately 9 a.m. the patient noted the onset of a severe right unilateral headache with paralysis of the left arm and leg. On admission to the hospital she was alert and cooperative. Examination revealed a left hemianesthesia and hemiplegia. The right pupil was dilated. A neurosurgical consultant's impression was intracranial hemorrhage. A ruptured cerebral aneurysm or hemorrhage secondary to metastatic tumor were the suggested etiology. In view of the patient's poor condition no additional studies were peformed. At approximately 2 a.m. the patient was noted to have a respiratory arrest. Caramine injections and artificial respiration failed to revive her. She was pronounced dead at 2:30 a.m. on December 11, 1956. An autopsy was performed on December 12, 1956 at 8:00 a.m.

Discussion

Benign moles are adequately treated by evacuation of the uterus. Following evacuation of the uterus the gonadotropic hormone excretion returns to negative within three months period in 96 per cent of cases. However, the persistence of an elevator titer does not necessarily indicate the presence of choriocarcinoma, *provided the titer does not rise*. The longest persistence of a positive titer was ten months in a series of 54 cases reported by Lane.[2]

Malignant mole or chorio-adenoma destruens is defined as invasive mole which *does not metastasize*. The malignant nature being invasion of the uterine wall and blood vessels without tissue destruction. The mortality in reported cases results primarily from perforation of the uterus with hemorrhage and sepsis. The continued trophoblastic activity results in continued gonadotropic hormone excretion as would be expected. With hysterectomy as the treatment of choice, a 100 per cent cure rate can be expected according to Hertig and Sheldon.[1] The diagnosis of malignant mole, however, is satisfactorily made only on examination of the hysterectomy specimen. Curettage is almost uniformly unreliable since choriocarcinoma and malignant mole are invasive in nature with the bulk of the tumors being within the uterine wall beyond the range of the curet.

It is generally agreed that choriocarcinoma is universally fatal although cures have been reported. Most authors agree that cures if reviewed critically would fall into the category of malignant mole or syncytioma (retained products of conception at the placental site following delivery or abortion). The diagnosis of choriocarcinoma depends upon (a) proof of distant metastasis, (b) increasing gonadotropic titer or (c) microscopic examination of the uterus. An intervening pregnancy will result in an increasing gonadotropic hormone excretion. Since early pregnancy is extremely difficult to rule out, the diagnosis then seems to depend entirely on the hysterectomy specimen or proof of distant metastasis.

In the case at hand the continued hemorrhage combined with high levels of gonadotropic hormone excretion for longer than six months seems to favor the diagnosis of either malignant mole or choriocarcinoma.

Since the possibility of an intervening pregnancy has been satisfactorily eliminated, hysterectomy was recommended. A diagnosis of benign mole could not be absolutely excluded. In view of this she desired to retain her child bearing function even though she was fully aware of the possible consequences. Until 11 days prior to her death the patient was well, without evidence of metastasis, eight months since passage of the mole.

At autopsy a 4.5 cm. hemorrhagic necrotic mass was found occupying the entire fundal portion of the endometrial cavity. There were no pelvic adhesions and no evidence of local metastasis. No abnormalities were noted in either adnexa. Examination of the lungs and liver failed to reveal any grossly or microscopically identifiable tumor nodules. The brain contained a large 8 x 5 x 3 cm. intracerebral hematoma in the right temperoparietal region which was surrounded by a light brown and gray halo of tissue grossly felt to represent tumor tissue. Microscopic examination revealed invasive trophoblastic cells within the uterine wall. Serial blocks from the brain tissue failed to reveal the etiology of the hemorrhage. Only hemorrhagic and necrotic brain tissue was present with no recognizable metastastic tumor cells. The microscopic sections were reviewed by numerous pathologists and a final diagnosis of choriocarcinoma with intracerebral hemorrhage probably secondary to metastasis was made.

Summary

1. A case of hydatidiform mole is presented with continued uterine hemorrhage and high levels of gonadotropic hormone excretion for longer than eight months following its passage.

2. A brief review of the diagnosis and treatment of benign mole, chorio-adenoma destruens and choriocarcinoma is presented.

3. Final diagnosis of choriocarcinoma was made by autopsy despite failure to definitely identify a metastastic lesion in the brain.

4. This report is presented with the hope that all practitioners of obstetrics may be made more aware of the difficulties associated with the diagnosis and management of these uncommon obstetrical conditions.

BIBLIOGRAPHY

1. Hertig, Arthur T., and Sheldon, Walter H. Hydatidiform mole—a Pathological-clinical correlation of 200 cases. American Journal of Obstetrics and Gynecology 53: 1: 1947.
2. Lane, F. L., Hormone studies in the presence of Hydatidiform mole and chorioepithelioma surgery, Gynecology and Obstetrics 73: 86: 1941.
3. Novak, Emil. Gynecological and Obstetrical Pathology. 3rd Edition p. 503.

ADDENDUM: I would like to express my thanks to James Pitts, M.D. for permitting me to use his private case for this presentation and to William T. Snoddy, M.D., Pathologist at St. Anthony's Hospital for his help in the formalization of this paper.

(601 Northwest 9th, Oklahoma City, Oklahoma)

PLAN TO ATTEND

OSMA ANNUAL MEETING

MAY 5, 6, 7

OKLAHOMA CITY

PRESIDENT'S LETTER

We are living in a most remarkable era of time. Our profession has witnessed more scientific advances in medicine, during the last fifty years, than had occurred in all previous recorded history. Science has brought many of our so called "empirical practices" into the fold of reason and explanation. Science has likewise directed our endeavors into new and unchartered areas of therapy, with a response on the part of many of our educators, of utter abandon of the individual or person.

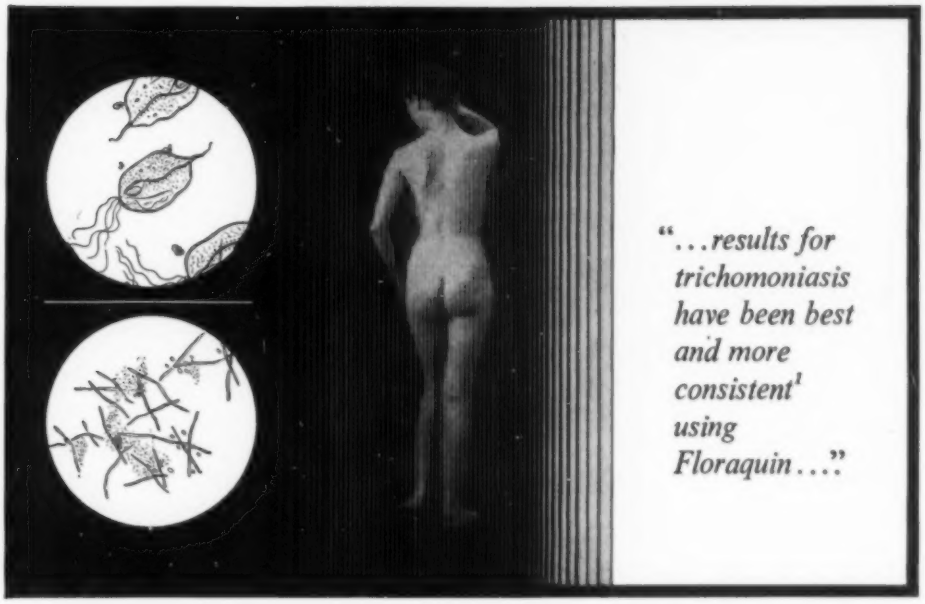
This pseudo-scientific cynicism is, to my mind, thoroughly un-becoming to the medical profession and behooves our criticism. We should always welcome improvement and advances in medicine, but we must at all times keep in mind we are applying these to human beings like ourselves. We should be reminded of Section One of the American Medical Association's Principles of Medical Ethics.

"The principal objective of the medical profession is to render service to humanity with full respect for the dignity of man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion."

John Flack Barton, M.D.

President

EFFECTIVE, DEPENDABLE THERAPY FOR VAGINITIS



Floraquin® eliminates trichomonal and mycotic infection; restores normal vaginal acidity

Leukorrhea is by far the most frequent symptom of vaginitis; trichomonads and monilia are the most common causes. Many authors have reported[2] trichomonal protozoa in the vagina of 25 per cent of obstetric and gynecologic patients. Increased use of broad spectrum antibiotics has resulted in a sharp rise in the incidence of monilial infections.

Floraquin effectively eradicates both trichomonal and monilial vaginal infections through the action of its Diodoquin® content. Floraquin also furnishes boric acid and sugar to restore the normal vaginal acidity which inhibits pathogens and favors the growth of protective Döderlein bacilli.

Pitt[1] recommends vaginal insufflation of Floraquin powder daily for three to five days, followed by acid douches and the daily insertion of Floraquin vaginal tablets throughout one or two menstrual cycles. G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

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1. Pitt, M. B.: Leukorrhea. Causes and Management, J. M. A. Alabama *25*:182 (Feb.) 1956.
2. Parker, R. T.; Jones, C. P., and Thomas, W. L.: Pruritus Vulvae, North Carolina M. J. *16*:570 (Dec.) 1955.

SEARLE

I take pleasure in introducing to the physicians of Oklahoma this new section in their Journal. *It is with self-warming pride that we of the Medical Center will lay before you in more or less synoptic fashion a picture of the work that we are doing. We trust that those of our people who are very close to the origin of medical knowledge will be able by this means to informally bring you useful information and a deeper understanding of the forces of nature expressed in disease that are being brought under control. Perhaps, in time, as we get better acquainted there can be an exchange of ideas relating to the philosophy of medical practice, medical education, medical service and medical research that will bury forever the ghost of a dichotomy that seems to hover over us.*

To the editors of the section I offer my best wishes and what help I can toward accomplishing the aims set out; to the editors of the Journal *my thanks for these pages; and to the readers I send my warmest regards.*

Sincerely,

Mark R. Everett

Director and Dean
University of Oklahoma
Medical Center

Pathogenesis of Herpes Simplex Virus*

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AUTHOR'S NOTE

L. Vernon Scott, Sc. D., received his Master of Science degree from the University of Oklahoma in 1947 and his Doctor of Science degree from Johns Hopkins University in 1950. His specialty is Virology. At the present time Doctor Scott is Associate Professor of Microbiology, at the University of Oklahoma School of Medicine.

Doctor Scott is a member of the Society of American Bacteriologists, the American Association for Advancement of Science, an Associate Member of the Society of Experimental Biology and Medicine, the Oklahoma Academy of Science, the New York Academy of Science, and the Sigma Xi.

The virus of herpes simplex, commonly called "fever blister" or "cold sore" virus, is one of the most prevalent infectious agents of man. Animals are infected only experimentally. It can be isolated in the laboratory with relative ease.[1]

The host responds to a primary infection with this virus as it does to any other infection with the production of antibodies which can be demonstrated by the usual serological tests, such as neutralization, complement fixation and hemagglutination.[2] According to Scott and colleagues[3] the primary attack may produce diseases of the skin, mucous membranes, eye and its enveloping tissues and central nervous system, as well as generalized infections.

Following a primary infection, recurrent attacks occur in persons who have demonstrable neutralizing antibodies. With the primary infection there is usually a systemic illness which may be quite severe and sometimes fatal. However, in only about 15 per cent of the people does the first invasion or primary infection lead to clinical symptoms. In about 85 per cent there are no clinical indications of infection with the virus. In these subclinical cases the only proof of the disease is the detection of antibodies in the blood. Individuals who have recovered from either a manifest or subclinical infection become carriers of this virus. Following nonspecific stimuli, such as shock, menstruation, emotional upsets, fever and unrelated infections, recurrent manifestations of this virus become apparent in the host. He then becomes a carrier of the virus and he can spread it to other susceptibles. The nature and site of the lesions produced influence the spread. The lesion is a sharply defined group of thin-walled vesicles on an erythematous base. These may occur on any area of the skin or mucous membranes with a predilection for the mucocutaneous junctions. The vesicular fluid contains the virus and when the lesions are located in the mucous membranes of the mouth the tops of the vesicles may be eroded away and thus the saliva is infectious.

This virus has been isolated from diseases of the skin known as recurrent herpes simplex (fever blister), eczema herpeticum (Kaposi's varicelliform eruption) and traumatic herpes. It has been found in acute gingivostomatitis, herpes labialis, herpes progenitalis and vulvovaginitis of the mucous membranes and mucocutaneous junction. Herpes simplex infections of the eye may appear as a conjunctivitis or more commonly as a keratoconjunctivitis and a meningoencephalitis develops when the virus invades the central nervous system. This virus has been isolated from generalized infections by Womack and Randall[4] and Foerster and Scott[5] from cases of erythema multiforme exudativum; by Morgan and Finland[6] from a case of atypical pneumonia and erythema multiforme exudativum; by Zuelzer and Stulberg[7] from a case of fulminating visceral disease and hepatitis in infancy and by Quilligan and Wilson[8] and McDougal and colleagues[9] from fatal infections in newborn infants.

In recent years increasing attention has

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been focused on the generalized effect of this virus, particularly in newborn infants. Germer[10] has postulated that this virus may be a cause of fetal and infant death. The pathogeneses of the diseases caused by this virus are not clear. The role of a viremia in the spread of the virus from the initial site of infection to various organs has not been carefully studied. The conditions responsible for the activation of latent infections and for the production of a viremia in recurrent infections where a certain amount of residual antibody is presumed to be present are still a mystery. The virus may be acquired from genital lesions during passage out the birth canal but this does not explain the pathogenesis of the disease in infants who are born with herpetic infections.

A study was initiated to determine the effect of herpes simplex virus upon the adult female rabbit and its fetuses. Before fetal experiments could be carried out it was necessary to establish a viremia in this animal and to determine the extent of it. After the establishment of a viremia, studies could be undertaken to determine if virus could get through the placenta to infect the fetuses.

The method of dissemination of herpes simplex virus from the site of initial infection in the rabbit to various organs where disease is manifested may be accomplished in several ways. According to van Rooyen and Rhodes,[11] Levaditi in 1922 claimed that this virus spread from the cornea of the rabbit by way of the retina and optic nerve and Goodpasture and Teague[12, 13] demonstrated inclusion bodies in sections of nerve tissue and in the endothelial cells of blood vessels following inoculation of the virus on the cornea of the rabbit. This was evidence that a route of dissemination of the virus was along the nerve tracts. However, they suggested that the blood may sometimes contain the virus. During the same year Marinesco and Draganesco[14] reported that this virus passed through the tissue spaces and vessel sheathes of nerve cells. Goodpasture[15, 16] continued to present evidence to support the idea that the herpes virus travelled along the axis cylinders of peripheral nerves and so entered the central nervous system pathways. Since viral isolation technics are now available a critical study of this mode of dissemination is indicated.

Field[17] in 1952 studied the pathogenesis of herpes simplex encephalitis following corneal inoculation. Certain histological findings were suggestive of axonic transmission. However, the "lymphatic space" of Marinesco was considered, as well as bloodborne methods of transmission. More recently Boyse and colleagues[18] used radioactive iodine labelled homologous plasma protein following injection of herpes simplex virus into nerves as a means of detection of virus spread. This protein preferentially accumulated at the sites of inflammation. From this study he presented evidence of virus dispersion through tissue fluids surrounding the nerves rather than along the nerve tracts. Thus, another method of transmission is suggested.

Early evidence to support the idea that the virus was spread from the initial site of infection to other areas by the blood was given by Doerr and Vöchting[19] in 1920 who were able to produce encephalitis in rabbits following intravenous injection of the virus. Greenbaum and Harkins[20] reported that concentrated blood from an infected rabbit could produce a typical keratoconjunctivitis in normal rabbits following corneal inoculation without producing an involvement of the central nervous system. Remlinger and Bailly[21] used whole blood, defibrinated blood and serum from experimentally infected rabbits as the specimens and succeeded in transmitting herpes simplex virus to other rabbits following intracerebral, intravenous and subcutaneous injections. Gildemeister and Heuer[22] in 1929 also demonstrated that the virus could be isolated from the blood of rabbits by animal inoculation and Kipping[23] who described generalized herpetic infections was of the opinion that the distribution of the lesions suggested a dissemination of the virus by the blood stream. Haymaker[24] presented pathological studies of herpetic encephalitis in humans in which he found intranuclear inclusion bodies in the brains. He suggested that the spread of the herpes simplex virus from the site of original infection to the central nervous

system could be by means of the blood stream. More recently Ruchman and Dodd[25] in 1950 recovered herpes simplex virus from the blood of a patient with herpetic rhinitis.

Earlier, Anderson[26] showed experimentally that at certain stages in the development of the chick embryo, the herpes simplex virus spreads to organs distant from the site of inoculation by way of the blood stream. She demonstrated that in such embryos the virus no longer showed its customary ectodermatropism but was capable of attacking mesodermal and entodermal structure with equal ease. This confirmed earlier observations of Goodpasture and Teague.[12]

Zuelzer and Stulberg[7] in describing cases of fulminating visceral herpetic infections and herpetic hepatitis postulated that the virus was spread from the focus of primary infection by the blood stream. However, Geller and colleagues[27] inoculated herpes simplex virus into the veins of human volunteers and was unable to unequivocally demonstrate the virus from either plasma or cellular elements as early as midway through the virus infusion.

Thus, it may be noted that from a survey of the literature the virus was spread 1) along the axons of nerve cells, 2) through the sheathes and fluids surrounding the neurons and 3) by hematogenous dissemination. Whether the virus spreads along the nerve cells or along perineural lymphatics is not known but early evidence involves the nerves and surrounding fluids as pathways of infection. Dissemination through the circulatory system may serve as a pathway of infection because intravenous injections may give rise to herpetic encephalitis. Experimentally, Marinesco and Draganesco[28] injected virus into the peripheral extremity of a cut sciatic nerve and herpetic encephalitis developed as a result of blood dissemination. Thus, it would appear that the virus is spread by a combination of methods.

If herpes simplex is present in the blood during infection then there is a possibility that the virus which is in the blood of pregnant mothers may get into the fetus and produce characteristic lesions.

Evidence that a virus could produce congenital anomalies was reported by Gregg and colleagues[29] in 1942 when they described the malformations in babies being delivered of mothers who had been infected with rubella during the first trimester of pregnancy. The principle defects noted were eye and heart disease, deaf-mutism and possible mental defectiveness. Since that time there have been many reports to substantiate this work. The rubella virus has not been isolated from infected fetuses of man or experimentally infected animals.

Belenky[30] reported the presence of the vaccinia virus in the blood and placenta of pregnant animals following vaccination but was unable to detect the virus in the amniotic fluid, umbilical cord, or in the organs and blood of the embryo. Hass[31] demonstrated that an inapparent infection resulted when mice acquired an infection with lymphocytic choriomenigitis *in utero* and they tended to retain active virus. Mice infected after birth had apparent infections and those that recovered were not carriers for long periods of time. They transmitted the infection to offspring and contacts. This is particularly interesting because of 1) the continuous propagation of an infection that is inapparent and 2) the basic difference in response to infection shown by *in utero* infection as compared to the response of mice subjected to infection after reaching maturity. Berry and Slavin[32] were able to isolate herpes simplex virus from the blood of infected adult mice but not from their embryos. Scheidegger[33] reported that the embryos of mice died when the adults were inoculated with psittacosis or ectromelia virus. Winsser and colleagues[34] discovered a viremia in a newborn human whose mother suffered paralytic poliomyelitis shortly before childbirth and Schaeffer and colleagues[35] isolated poliovirus from the aborted fetus of a mother who had the disease. Quilligan and Wilson[8] and Williams and Jack[36] described fatal herpes simplex infections in newborn with typical skin and liver infections.

In view of the re-evaluation of the pathogenesis of poliomyelitis upon the detection of a viremia stage in that disease by Bodian[37] and Horstmann[38] this study was undertaken. It was designed to determine if

herpes simplex virus could be demonstrated in the blood of the rabbit at varying hours after inoculation of the virus on the scarified cornea and, if so, then to further determine if the virus could pass through the placenta of an infected, pregant rabbit during the viremia stage and invade the fetal tissues.

Materials and Methods

Virus: The HF strain of herpes simplex virus, obtained from the Communicable Disease Center in Montgomery, Alabama was used. It had been propagated 60 times on the chorioallantoic membranes (CAM) and nine times in the yolk sacks (YS) of the developing chick embryos, three times in the brains of white Swiss mice (MB) and two times on the corneas of rabbits. The seed virus was inoculated into the YS of the chick embryo and the resulting infected allantoic fluid was harvested and used as the inoculum. The LD_{50} of the viral inoculum as determined by the YS method was greater than 10^5 per ml.

Animal inoculation: Adult white rabbits which are highly susceptible to ocular infection with herpes simplex virus were lightly anesthetized and the corneas of both eyes were scarified, deep enough to penetrate the corneal epithelium, with sterile wire gauze. One-tenth ml. of a homogenous suspension of virus containing at least 10^4 ID_{50} for YS was placed in the left eye and an equal volume of sterile diluent was placed in the right eye. The eyelids were massaged over the corneas to rub the inocula into the scarified areas. Either sterile skimmed milk or buffered gelatin saline was used as the diluent. All diluents contained 250 units of penicillin and 250 micrograms of streptomycin per ml. These same diluents were used in the preparation of the respective specimens for inoculation onto the CAM and into the YS and brains of mice.

Virus studies in adult rabbits: One ml. of blood was removed aseptically from the marginal ear veins of the rabbits at specified times. Ocular washings were obtained from each rabbit 48 hours after corneal inoculation. Autopsies were performed on rabbits that died and specimens of brain, heart, heart blood, liver, kidney and spleen were obtained. Rectal temperatures were recorded prior to inoculation and at the time each specimen of blood was taken.

The presence of the virus was determined by the inoculation of prepared specimens of blood, ocular washings and tissue homogenates of autopsy organs onto the CAM. Two dilutions of each specimen were used (1:2 and 1:20) and 0.1 ml. of each dilution was inoculated onto the CAM of the developing chick embryo according to the method of Scott[1] with minor modifications. The embryos were incubated 72-96 hours at 36° C to allow for virus proliferation. The inoculated areas of the CAM were removed and the presence of the virus was determined by the production of plaques on each membrane. Six embryos were used for each dilution of each specimen. When results were doubtful with the first inoculated CAM they were homogenized and prepared as the specimens and reinoculated into a second group of embryos. To confirm the results obtained by the CAM inoculations, selected specimens were inoculated into the YS and the MB. Virus neutralization technics were used to identify the isolated virus.

As controls, a group of developing chick embryos was inoculated via the CAM route with appropriate dilutions of 1) normal blood and buffered gelatin saline, 2) normal blood and sterile skimmed milk, 3) normal blood, buffered gelatin saline and virus and 4) normal blood, sterile skimmed milk and virus. These were treated in the same manner as the rabbit specimens. A portion of the initial rabbit inoculum was placed onto the CAM of the chick embryo and intracerebrally into mice to demonstrate infectivity.

Fetal studies: Fourteen days after breeding, six young adult white female rabbits were lightly anesthetized with ether and 0.1 ml. of the virus suspension was massaged into the scarified cornea. One ml. of blood was removed aspetically from the marginal ear veins of the rabbits at 24, 48 and 54 (just prior to surgery) hours and every 12 hours thereafter through 180 hours. The presence of the virus in these samples, as well as in 48 hour ocular specimens was determined by the production of lesions on the CAM.

Fifty-four hours following inoculation, the rabbits were anesthetized with ether and surgical procedures were used to remove one gravid horn of the bifurcate uterus. The other horn was left intact so that the rabbit might go to term and deliver.

The fetuses from each rabbit were dissected from the excised uterine horns, washed thoroughly with sterile physiological saline and ground in sterile tissue grinders. This material was diluted 1:2 with buffered gelatin saline and 0.1 ml. of this homogenate of fetuses from each rabbit was inoculated onto each of eight CAM. The inoculated embryos were incubated for 72 hours. At this time the membranes were harvested and examined for typical herpes simplex plaques which would indicate the presence of the virus in fetal tissue.

Results

Virus studies in adult animals: Herpes simplex virus was isolated from the blood of rabbits at varying times following inoculation of this virus on the scarified cornea as seen in Table 1. It was found as early as 24 hours and as late as 180 hours after inoculation. Positive blood specimens were obtained from each rabbit which had positive corneal infections. However, not all blood specimens were positive at the designated hours. The 24, 48, 54, 60 and 96 hour specimens appear to be the optimal times for the demonstration of the virus. No virus was noted in the 78 and 102 hour samples.

The virus was cultured from the ocular washings of the infected eye of each rabbit 48 hours after inoculation. This eye and its enveloping membranes exhibited severe swelling, redness, and injection with multiple vesicles on the cornea and membranes. Later the cornea became cloudy. Specimens taken from the control eye which appeared essentially normal yielded no virus.

In most cases, the rectal temperatures of the rabbits rose steeply from the normal during the 48 to 108 hour period and then returned to normal in those rabbits that did not die. The temperatures taken just before death were sub-normal.

The virus was isolated from certain organs upon death of the animal as shown in Table 2. The organs from which the virus was sought were brain, heart blood, heart, kidney, liver, lung and spleen. All animals that were autopsied exhibited central nervous system involvement resulting in convulsions prior to death. The virus was not isolated from all specimens of all organs from each rabbit. However, part of the samples from each rabbit contained virus. Rabbit 10 appeared healthy three months after inoculation. This animal developed symptoms of central nervous system involvement and died after receiving subcutaneous injections of formalin inactivated mouse brain propagated herpes simplex virus. Virus was isolated from five autopsy organ specimens. Rabbit 25 apparently recovered from the initial infection of the cornea and viremia and 14 days after inoculation delivered three offsprings. At this time she developed a severe recurrent herpetic conjunctivitis in the previously infected eye and died 48 hours later after exihibiting central nervous system involvement. Virus was isolated from the eye, brain and liver of this animal.

Fetal studies: Herpes simplex virus was isolated from the fetuses of six rabbits which had been infected 54 hours prior to surgical removal of the embryos. The virus was also isolated from the ocular washing and blood samples (24, 48 and 54 hours) of these animals. The adult rabbits were 14 days pregnant when inoculated. The isolated virus was identified as herpes simplex virus

Viremia in Rabbits after Corneal Inoculation

Hours at which blood samples were taken	24	48	54	60	66	72	78	84	90	96	102	108	120	132	144	156	168	180
Viral isolations	16	16	12	13	4	5	0	8	1	13	0	8	6	7	5	3	3	4
No. isolations attempted	22	22	13	16	5	16	2	17	5	17	4	11	11	11	6	6	5	5

Table 1

Fate of Animals and Presence of Virus in Organs after Autopsy

Rabbit	Elapsed time between inoculation and death	Presence of virus in organs						
		Brain	Heart Blood	Heart	Kidney	Liver	Lung	Spleen
1	96 hours	+	+	—	—	+	—	+
3	21 days	+	+	U	U	U	U	U
10	3 mos.	+	+	—	—	+	+	+
13	21 days	+	+	+	+	+	+	U
14	23 days	+	+	+	+	+	+	+
19	156 hours	+	U	—	U	+	+	+
25	14 days	+	U	U	U	+	—	U

\+ Virus isolated on CAM
— Virus not isolated on CAM
U Isolations not attempted

Table 2

by the neutralization technic. Table 3 shows a comparative titration of this virus in the presence of specific immune serum and in buffered gelatin saline.

Discussion

The demonstration of the virus in the blood of rabbits after establishment of corneal infection with herpes simplex virus proves conclusively that the virus can be present in the blood during certain stages of the disease. The isolation of virus from the various organs indicated the wide dissemination of the virus. Even though the tissues were thoroughly washed it is not known whether the virus which was isolated from the tissues came from virus in the blood or from foci of infection in the tissues.

It is interesting to note that virus was isolated as late as 180 hours (nearly eight days) after infection. It has been shown by Scott and colleagues[3] that following the primary infection in children the circulating neutralizing antibodies against the virus appear in the blood as early as the fourth day of illness and rise steeply. The majority of the 31 children in his study had a neutralizing titer of 1:16 by the eighth day after onset. Perhaps the reason the viremia endpoint was not reached by the eighth day in the rabbits was that the time of inoculation was used as the beginning time for experimental work on rabbits and the work of Scott and colleagues[3] used the onset of primary infection as the beginning time. There is an incubation period of three to five days in human beings which could account for this difference. Experiments to correlate the presence of virus in blood and the production of neutralizing antibodies are being done.

The death of rabbit 10 during vaccination three months after initial infection is of interest for two reasons. First, infectivity of the vaccine for CAM could not be demonstrated however, herpes simplex virus was isolated from the brain of the rabbit at autopsy, indicating a latent virus might have been activated. Good and Campbell[39] demonstrated that a latent herpes simplex encephalitis could be activated by the production of anaphalactic shock in their animals. The immunization procedure in rabbit 10 had been in progress for three weeks. It consisted of the subcutaneous inoculation of mouse brain which contained the virus. The amount of mouse brain protein in ration to virus protein was extremely high. Therefore, a hypersensitivity reaction produced by the immunization procedure may have been the mechanism which triggered the recurrent attack. Second, there should have been residual antibodies present to prevent this recurrent attack. Following a primary attack in humans a certain level of neutralizing antibodies remained for many months according to Jawetz and Coleman.[40] However, Wenner[41] noted a complete disappearance of antibody from the blood of a

Comparative Titration of Virus Isolated from Fetuses

Inoculum	Dilution of virus							
	undiluted	10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}	10^{-6}	10^{-7}
Virus-antiserum mixture	++++	++++	+++	++	++	+	—	—
Virus-saline mixture	++++	++++	++++	+++	+++	+++	++	+

++++ CAM havily infected, plaques too numerous to count
+++ 16-25 plaques on CAM
++ 15-6 plaques on CAM
+ 1-5 plaques on membrane
— no plaques on membranes

Table 3

20 month old infant about seven months after recovery from a primary attack of eczema herpeticum. Antibody studies were not done on this rabbit therefore, it is not known if any residual antibodies were present to neutralize the virus.

Rabbit 25 experienced a recurrent attack at the birth of her offsprings. Whether the killing attack was a continuation of the primary attack or a recurrent attack of a latent virus is not known because viremia studies were not continued throughout the fourteen days. However, the keratoconjunctivitis had subsided and neutralizing antibodies should have been formed to prevent the recurrent attack.

The isolation of herpes simplex from the fetuses of rabbits does not prove that the virus was being proliferated there however, it probably was. In the adult the viremia could be due to sporadic showering of the virus into the blood from the foci of primary infection. However, in the fetus it probably is different. The virus could have been transported to the placenta by the maternal circulation where it entered the fetus and a new foci of infection might have been established. How the virus got across the "placental barrier" and into the fetus is a good question.

It is not surprising that the virus was proliferated in embryonic tissue. It has been well established that this tissue is more susceptible than adult tissue. Anderson[26] demonstrated the special susceptibility of chick embryo to this virus and Kilbourne and Horsfall[42] showed that newborn mice were more susceptible than older animals.

The role of passive antibody in the protection of human fetuses has not been established. There is ample proof that about 93 per cent of the people in the lower income brackets and 37 per cent in the high income brackets have demonstrable neutralizing antibodies to this virus following primary infection. It is presumed that these antibodies would be passively transferred and protect the susceptible fetus. There is clinical proof that the newborn humans have become infected during passage out the birth canal from mothers who were suffering genital infections. In these cases the passively acquired antibody was not effective in the prevention of the neonatal infection and these children were liable to a special risk. Zuelzer and Stulberg[7] were of the opinion that maternal antibodies are not adequate for the protection of premature infants, probably because of a special susceptibility of their tissue. It seems probable that a viremia is a frequent happening in primary herpetic infections in infants. The neutralizing antibodies are presumed to be effective in the prevention of a viremia in humans during a recurrent infection. In the primary infection the visceral lesions which are seen in infants are seldom experienced in older children. It is assumed that if specific immunity is lacking in both groups a non specific maturation of tissue, in analogy with the animal experiments, could be the answer for the difference in susceptibility of the two age groups.

If in pregnant female adults who do not have sufficient protective antibodies, a viremia were to develop during the recurrent infection then it is possible that a trans-

placental infection might occur. Whether the herpes simplex virus can cause fetal malformations in a manner similar to rubella is not known but it is possible that damage to differentiating embryonic tissue could take place. There is no evidence in human beings that a carrier state develops *in utero* without demonstrable neutralizing antibodies as in mice with lymphocytic choriomeningitis.

Summary

Herpes simplex virus was isolated from the blood, certain organs and the fetuses of adult white rabbits following inoculation of the virus onto the scarified cornea. The virus could be detected in the blood as early as 24 and as late as 180 hours following inoculation. The virus was demonstrated in the specimens by the production of herpetic plaques following inoculation of the specimens onto the chorioallantoic membranes of the developing chick embryo and was identified by specific neutralization procedures.

Recurrent infections were observed in two rabbits, one following an immunization procedure and another following birth of offsprings. The possible triggering mechanisms are discussed.

The possible role of a viremia in fetal infections in human beings, the relationship of maternal antibodies to fetal and neonatal infection, the susceptibility of fetal tissue to viral infection and the possibility of fetal malformations due to viral infection are discussed.

REFERENCES

1. Scott, T. F. M.: Chapter on Herpes Simplex in "Diagnostic Procedures for Virus and Rickettsial Diseases." pp. 313-340. Published by American Public Health Association, New York City, 1956.

2. Scott, L. V., Felton, F. G., and Barney, J. A.: Hemagglutination with Herpes Simplex Virus. J. Immunol. 78: 211-213, 1957.

3. Scott, T. F. M., Coriell, L., Blank, H., and Burgoon, C. F.: Some Comments on Herpetic Infection in Children with Special Emphasis on Unusual Clinical Manifestations. J. Pediat. 41: 835-843, 1952.

4. Womack, C. R., and Randall, C. C.: Erythema Exudativum Multiforme, Its Association with Viral Infections. Amer. J. Med. 15: 633-644, 1953.

5. Foerster, D. W., and Scott, L. V.: Isolation of Herpes Simplex Virus from a Patient witn Erythema Multiforme Exudativum (Stevens-Johnson Syndrome). To be Published.

6. Morgan, H. R., and Finland, M.: Isolation of Herpes Virus from a Case of Atypical Pneumonia and Erythema Multiforme Exudativum. Amer. J. Med. Sci. 217: 92-95, 1949.

7. Zuelzer, W. W., and Stulberg, C. S.: Herpes Simplex Virus as the Cause of Fulminating Visceral Disease and Hepatitis in Infancy. Amer. J. Dis. Child. 83: 421-439, 1952.

8. Quilligan, J. J., Jr., and Wilson, J. L.: Fatal Herpes Simplex Infection in Newborn Infant. J. Lab. & Clin. Med. 38: 742-746, 1951.

9. McDougal, R. A. Beamer, P. R., and Hellerstein, S.: Fatal Herpes Simplex Hepatitis in a New Born Infant. Amer. J. Clin. Path. 24: 1250-1258, 1954.

10. Germer, W.: Virus Infections as a Cause of Foetal and Infant Mortality. German Med. Monthly. 1: 61, 1956.

11. van Rooyen, C. E., and Rhodes, A. J.: Chapter on Herpes Febrilis in "Virus Diseases of Man," pp. 169-200. Published by Thomas Nelson & Sons, New York, 1948.

12. Goodpasture, E. W., and Teague, O.: Experimental Production of Herpetic Lesions in Organs and Tissues of the Rabbit. J. Med. Res. 44: 121-138, 1923-24.

13. Goodpasture, E. W., and Teague, O.: Transmission of the Virus of Herpes Febrilis along Nerves in Experimentally Infected Rabbits. J. Med. Res. 44: 139-184, 1923-24.

14. Marinesco, G., and Draganesco, S.: Recherches Experimentales sur le Neurotropisme du Virus Herepetique. Ann. Inst. Pasteur 37: 753-783, 1923.

15. Goodpasture, E. W.: The Axis Cylinders of Peripheral Nerves as Portals of Entry to the Central Nervous System for the Virus of Herpes Simplex in Experimentally Infected Rabbits. Amer. J. Path. 1: 11-28, 1925.

16. Goodpasture, E. W.: The Pathways of Infection of the Central Nervous System in Herpetic Encephalitis of Rabbits Contracted by Contact, with a Comparative Comment on Medullary Lesions in a Case of Human Poliomyelitis. Amer. J. Path. 1: 29-46, 1925.

17. Field, E. J.: Pathogenesis of Herpetic Encephalitis Following Corneal and Masseteric Inoculation. J. Path. & Bact. 64: 1-11, 1952.

18. Boyse, E. A., Morgan, R. S., Pearson, J. D., and Wright, G. P.: The Spread of a Neurotropic Strain of Herpes Virus in the Cerebrospinal Axis of Rabbits. Brit. J. Exper. Path. 37: 333-342, 1956.

19. Doerr, R., and Vochting, K.: Sur le Virus de l'herpes febrile. Rev. Gen. Ophthal., Paris. 34: 409, 1956.

20. Greenbaum, S. S., and Harkins, M. J.: Experimental Studies on Immunity to Herpes Simplex. Arch. Derm. and Syph. 11: 789-803, 1925.

21. Remlinger, P., and Bailly, J.: Diffusibility of Herpes Simplex Virus and Blood in Herpetic Encephalitis of Rabbits. Compt. Rend. Soc. Biol., Paris. 94: 109-114, 1926.

22. Gildemeister, E. and Heuer, G.: Ueber den Nachweis von Herpesvirus im Blute bei Experimentall Infizierten Kaninchen und uber Experimentellon Hautherpes. Dtsch. Med. Wschr. 55: 905-907, 1929.

23. Kipping, R. H., and Downie, A. W.: Generalized Infection with the Virus of Herpes Simplex. Brit. Med. J. 1: 247-249, 1948.

24. Haymaker, W.: Herpes Simplex Encephalitis in Man. J. Neuropath. & Exper. Neurol. 8: 132-154, 1949.

25. Ruchman, I., and Dodd, K.: Recovery of Herpes Simplex Virus from Blood of Patient with Herpetic Rhinitis. J. Lab. & Clin. Med. 35: 434-439, 1950.

26. Anderson, K.: Pathogenesis of Herpes Simplex Virus Infection in Chick Embryo. Amer. J. Path. 16: 137-155, 1940.

27. Geller, P., Coleman, V. R., and Jawetz, E.: Studies on Herpes Simplex Virus, The Fate of Viable Herpes Simplex Virus Administered Intravenously. J. Immunol. 71: 410-418, 1953.

28. Marinesco, G., and Draganesco, S.: Contribution a la Pathogenie et a la Physiologie Pathologique du Zona Zoster. Rev. Neurol. 39: 30-45, 1923.

29. Gregg, N. M., Beavis, W. R., Heseltine, M., Machin, A. E., and Vickery, D.: The Occurrence of Congenital Defects in Children Following Maternal Rubella During Pregnancy. Med. J. Austral. 2: 122-126, 1945.

30. Belenky (Belen'kii), D. E.: K voprosu o perekhode ospennago virusa cherez platsentu. J. Eksper. Biol. i Med. (Russia) 13: 30-36 (German summary p 36), 1929, abstracted, Biol. Absts. 8: No. 15897, 1934.

31. Haas, V. H.: Studies on the Natural History of Lymphocytic Choriomeningitis in Mice. Pub. Hlth. Repts. 56: 285-292, 1941.

32. Berry, G. P., and Slavin, H. B.: Studies on Herpetic Infection in Mice. J. Exper. Med. 78: 305-313, 1943.

33. Scheidegger, S.: Experimental Viral Infections in the Embryo and Fetus. Amer. J. Path. 29: 185-198, 1953.

34. Winsser, J., Pfaff, M. L., Seanor, H. E.: Poliomyelitis Viremia in a Newborn Infant. Pediatrics. 20: 458-467, 1957.

35. Schaeffer, M., Fox, M. J., Li. C. P.: Intrauterine Poliomyelitis Infection. J. Amer. Med. Assoc. 155: 248-250, 1954.

36. Williams, A., and Jack, I: Hepatic Necrosis in Neonatal Herpes Simplex Infection. Med. J. Austral. 1: 392-394, 1955.

37. Bodian, D.: A Reconsideration of Pathogenesis of Poliomyelitis. Amer. J. Hyg. 55: 414-438, 1952.

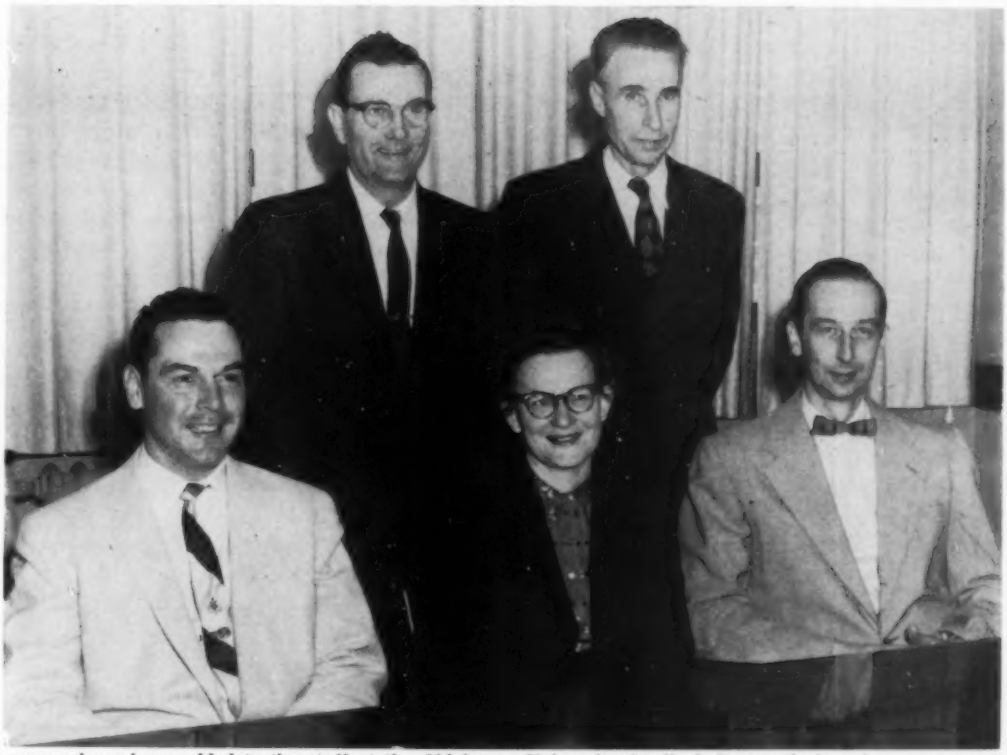
38. Horstmann, D. M.: Poliomyelitis Virus in Blood of Orally Infected Monkeys and Chimpanzees. Proc. Soc. Exptl. Biol. Med. 79: 417-419, 1952.

39. Good, R. A. and Campbell, B.: The Precipitation of Latent Herpes Simplex Encephalitis by Anaphylatic Shock. Proc. Soc. Exp. Biol. and Med. 68: 82-87, 1948.

40. Jawetz, E., and Coleman, V. R.: Studies on Herpes Simplex. IV. the Level of Neutralizing Antibodies in Human Sera. J. Immunol. 68: 655-661, 1952.

41. Wenner, H. A.: Complications of Infantile Exzema Caused by the Virus of Herpes Simplex. Amer. J. Dis. Child. 67: 247-264, 1944.

42. Kilbourne, E. D., and Horsfall, F. L.: Studies of Herpes Simplex Virus in Newborn Mice. J. Immunol. 67: 321-329, 1951.



Six names have been added to the staff at the Oklahoma University Medical Center during the current school year (left to right): Seated—Raymond Bower, Ph. D., (Microbiology), Miss Edna Schmidt, B.A., (Physical Therapy), M. J. Keyl, Ph. D., (Physiology); standing—John E. Allison, Ph. D., and L. G. Gumbreck, Ph. D., (both of Anatomy). McWilson Warren, Ph. D., (Preventive Medicine and Public Health) is not pictured.

New Members of the Faculty

John E. Allison, Ph.D., Associate Professor of Anatomy. Doctor Allison received his B.A. from Concordia College, Moorhead, Minnesota, in 1940; his M.A. from the University of Minnesota in 1947, and his Ph.D. from the State University of Iowa in 1952. His teaching experience includes: Instructor of Biology, Drake University, 1947-49. Instructor in Anatomy, St. Louis University School of Medicine, 1952-54, and Assistant Professor of Anatomy (at St. Louis), 1954-57. His research is concerned with the autonomic nervous system.

Raymond K. Bower, Ph.D., Research Associate, Department of Microbiology. Doctor Bower received his A.B. from the University of Kansas City in 1949 and his Ph.D. from Kansas State College at Manhattan in 1954. He completed a research fellowship at the University of Kansas School of Medicine under Doctor Herbert A. Wenner (Department of Pediatrics, Section for Virus Research). Doctor Bower was Director of the Virus and Rickettsia Division, Indiana State Board of Health. His research interests include: genetics of Newcastle disease virus and studies on tumor-inducing viruses.

Lawrence G. Gumbreck, Ph.D., Assistant Professor of Anatomy. Doctor Gumbreck received his B.A. from the University of Wisconsin in 1927 and his M.A. from Wisconsin in 1929. He was engaged in agricultural work from 1930-37, and returned to academic and scientific pursuits in 1938. Doctor Gumbreck was employed as an entomologist in state and city boards of health in Wisconsin in 1940, 1941 and 1944. He received his Ph.D. degree from the University of Wisconsin in 1945 with a major in Zoology and a minor in Medical Physiology. Doctor Gumbreck was an endocrinologist at Difco Laboratories, Detroit, from 1945 to

1949, and then became Research Associate in Surgery and Lecturer in Zoology at Washington University, St. Louis, from 1949 until 1957. His chief field of professional work and interest is endocrinology.

M. J. Keyl, Ph.D., Assistant Professor of Physiology. Doctor Keyl attended the University of Illinois, Valparaiso University and Idaho State (Navy V-12) and received his B.S. (1947) and M.S. in 1948 in Physiology from the University of Cincinnati. He was employed as Assistant Pharmacologist with the Wm. S. Merrell Co., Cincinnati until 1953. Doctor Keyl received his Ph.D. in Medical Physiology from Cincinnati in 1957. His thesis: "The Distribution and Some Physiological Properties of Naturally-Occurring Choline Esters." During the last year, Doctor Keyl has been studying the renal excretion of citric acid.

Edna Schmidt, B.A., Instructor in the School of Physical Therapy. Miss Schmidt received her B.S. degree from Texas State College for Women at Denton and took physical therapy training at Walter Reed Hospital, Washington, D.C. She received additional training at O'Reilly General Hospital, Springfield, Missouri, where she also taught part-time at the Physical Therapy School. Miss Schmidt was attached to the 36th General Hospital during World War II through campaigns in Africa, Italy and France. She then served as Physical Therapist at St. Joseph's Hospital, South Bend, Indiana, and received her M.Ed., degree from Marquette University, Milwaukee, in 1955.

McWilson Warren, Ph.D., Assistant Professor of Preventive Medicine and Public Health. Doctor Warren received his B.A. degree from the University of North Carolina in 1951, his M.S.P.H. from the University of North Carolina School of Public Health in 1952, and his Ph.D. from Rice Institute in 1957. His research interests include: parasite physiology, fat metabolism and the fate and uptake of carbohydrates in cestodes; physiological responses of parasite to host strain variations, and aerobic vs. anaerobic metabolism in adult cestodes.

VA Appointments

Alvin W. Paulson, M.D., was recently appointed to the Oklahoma City VA Hospital Staff as Chief of the Out-patient Service. He was graduated from the Washington University School of Medicine in St. Louis, Missouri, in 1932, served his internship at the University Hospital in Oklahoma City, and was a resident for one year in pathology in the same institution. He was in general practice in Clinton, Oklahoma, from 1934 to 1940. Doctor Paulson served in the U.S. Army from September 1940 to December 1945, and again from November 1947 to 1949. He joined the VA in April 1949 and was appointed Assistant Chief Medical Officer of the VA Regional Office in Oklahoma City. He left Oklahoma City in February 1955 to take a position as Chief Medical Officer of the VA Regional Office in Lubbock, Texas. Doctor Paulson succeeded Doctor Chas. W. Robinson as Chief of the Outpatient Service of the Oklahoma City VA Hospital, when Doctor Robinson retired after 38 years of service. Doctor Paulson is responsible for the supervision of all outpatient treatment furnished in this hospital to veterans with service-connected disabilities.

Jay T. Shurley, M.D., was recently appointed as Chief of the Neuropsychiatic Service at the VA Hospital in Oklahoma City, and as a Professor of Psychiatry, Neurology and Behavioral Sciences. He came to the Oklahoma City Hospital from the National Institute of Mental Health, Bethesda, Maryland, where he was a research section head. Doctor Shurley is a native of Texas and a graduate of the University of Texas School of Medicine in 1942. After serving an internship in Indianapolis he served a residency at Pennsylvania Hospital in Philadelphia as a Rockefeller fellow in psychiatry. He was in private practice of psychiatry and psychoanalysis in Philadelphia from 1947 to 1951, teaching at Jeffer-

son Medical College during the same period. In 1951 he became a member of the Philadelphia Association for Psychoanalysis. He returned to Texas in 1951 to go into private practice in Austin. He also was a lecturer in psychopathology at the University of Texas School of Social Work. Inducted in the Army in 1952, he spent the next two years as a Medical Corps Captain, teaching psychiatry at the Medical Field Service School in Fort Sam Houston. Doctor Shurley went to the National Institute of Health in 1954 as Acting Chief of the Adult Psychiatry Branch of Clinical Investigation. He worked with Doctor John C. Lilly on a project on "human isolation" which with new techniques demonstrated the individual's intense need for some kind of stimulation from the environment. This isolation project is still under way at Bethesda and Doctor Shurley is in the process of developing a similar project at the Oklahoma City VA Hospital.

Hugh B. O'Neil, M.D., recently joined the staff of the Oklahoma City VA Hospital as Chief of the Neurology Section. He also holds an appointment as Assistant Professor of Medicine, Psychiatry, Neurology, and Behavioral Sciences. He is uniquely qualified in that he is certified by the American Board of Internal Medicine and is also qualified in Neurology.

He graduated from Northwestern Medical School and had his medical residency at Cook County Hospital, Chicago, Illinois. He served as a Major in the U.S. Army from 1941-45. He had three years residency in Neurology at the Denver VA Hospital, and was previously an Instructor at Northwestern. His arrival has permitted the development of the first training program in Neurology in Oklahoma.

Roy W. Teed, M.D., was recently appointed to the Oklahoma City VA Hospital staff as Chief of the Ophthalmology Section of the Surgical Service. Prior to his coming to Oklahoma City he was Chief of the Ophthalmology Section of the Surgical Service of the VA Hospital in Dayton, Ohio. Doctor Teed was graduated from Middlesex University Medical School, Cambridge, Massachusetts, in 1935 and interned at the Middlesex Hospital for one year. Doctor Teed was in private practice from January 1937 to 1952. During this time he spent three years in the U.S. Army as a medical officer. From 1952 to 1955 he received his residency training in ophthalmology at Gill Memorial Hospital, Roanoke, Virginia, and at the University Hospital, Columbus, Ohio. He joined the VA in September 1955 at the VA Hospital, Dayton, Ohio. Doctor Teed is is member of the American Medical Association, the Massachusetts Medical Society, and the American Society for Research in Ophthalmology. He also holds an appointment as Instructor in Ophthalmology at the University of Oklahoma.

ABSTRACTS

THE FEATURES AND SIGNIFICANCE OF HYPERTROPHIC OSTEOARTHROPY

J. F. HAMMARSTEN* and J. O'LEARY**

A.M.A. Archives of Internal Medicine 99:431-441, March 1957

The roentgenologic appearance of onionskin layers or a thin line of new bone along the distal portions of the long bones is characteristic of hypertrophic osteoarthropathy, a disorder that takes it name from the enlargement of the bones seen in the later stages. The authors review observations on 22 patients with x-ray changes indicating hypertrophic osteoarthropathy. All had pulmonary lesions, which were malignant in all but two. Six patients had adenocarcinoma, primary in the lung, and an equal number had squamous cell carcinoma of the lung. Three had fibrosarcoma of the lung. One patient was seen with each of the following diagnosis: Alveolar carcinoma of lung, small cell carcinoma of lung, undifferentiated carcinoma of the lung, presumptive carcinoma of lung with clumps of malignant cells seen in sputum (operation or autopsy not performed), Hodgkin's disease involving the lung and adrenals, diffuse pulmonary fibrosis, and infected pulmonary infarction. The age distribution ranged from 24 to 83, with a mean of 59, and a preponderance in the sixth and seventh decades. There were 21 men and one woman. The correct diagnosis was made after the first examination in only three of the 22 patients; many were seen by several physicians before the true nature of the disease was recognized.

The most frequent diagnosis applied to the joint disease was rheumatoid arthritis. Diagnosis of Paget's

disease and acromegaly were considered in several patients. The pulmonary disease was most often overlooked or considered to be tuberculosis, cyst, or unresolved pneumonia. Gynecomastia was observed in 10 of the 21 men. Eight of the patients had acromegaloid facies, with prominent nose and furrowed brow. Some showed a striking resemblance to one another. It is noteworthy that neither of the patients with benign lung disease had joint pains, whereas 19 of the 20 with pulmonary malignancies had such pains. Clubbing occurred in all but one patient but was usually not noticed by the patient until his attention was directed to the sign. A brawny edema of the legs and periarticular swelling was found in 19 instances. Most of the patients (17) showed a diffuse thickness of the legs or forearms or both. Seventeen patients had joint symptoms as the first evidence of disease; they preceded the chest symptoms usually by more than 18 months. The chest x-ray films were not considered suggestive of malignant disease in three of the 20 patients with neoplasms. Bronchoscopy indicated the diagnosis in only two of the 15 patients with carcinoma who were subjected to this procedure. Examination of the sputum for malignant cells showed such cells in nine of 15 patients. Of the 14 tumors in patients who had thoracotomies, nine were resectable. All in whom the primary tumor was resected showed dramatic relief of symptoms of joints and extremities. The authors conclude that pulmonary neoplasms are the chief cause of hypertrophic osteoarthropathy in this part of the world.

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COMPARISON OF THE HYPERGLYCEMIC EFFECTS OF GLUCOCORTICOIDS IN HUMAN BEINGS

KELLY M. WEST*

Diabetes 6, No. 2:168-175, March-April, 1957

The carbohydrate metabolism regulating (or hyperglycemic) potency of a steroid may be quickly and easily estimated by testing the degree to which it modifies glucose tolerance (as measured by a single blood glucose determination two hours after glucose by mouth). The hyperglycemic potency of a steroid may be accurately estimated by testing only a few subjects if they are tested to both a known and an unknown steroid with similar characteristics of time-action.

Using such a method the carbohydrate metabolism regulating potencies of oral prednisone and prednisolone were measured and no significant difference found.

Under the circumstances of this experiment prednisone and prednisolone were more than five times more potent than cortisone acetate in their hyperglycemic effects. Prednisone and hydrocortisone were identical in effect when administered orally in a dosage ratio of one to four.

Older subjects showed a "positive" response to this steroid-glucose test more frequently than younger.

The strikingly similar responses of identical twins to glucose tolerance tests and to steroid-glucose tolerance tests suggest that heredity plays a significant role in conditioning the hyperglycemic response both to glucose loading and to glucocorticoids.

Nondiabetic subjects with a family history of diabetes showed significantly greater hyperglycemic responses to prednisone and prednisolone-glucose tolerance tests than a control group.

The responses to prednisone-glucose tolerance testing of fourteen subjects with an intensely positive family history (both parents diabetic) were no greater than the responses of subjects with a much less significant family history. These latter data are not adequate to permit a final conclusion but suggest that the steroid-glucose tolerance test has only a limited ability to predict the eventual development of diabetes.

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USE OF RADIOISOTOPES IN THE DIAGNOSIS OF ANEMIA

PHILIP C. JOHNSON,* ROBERT M. BIRD,** and WILLIAM L. HUGHES.***

A.M.A. Arch. Int. Med. 100:544, 1957

Two radioisotope techniques are useful in the clinical diagnosis of anemia by allowing one to separate hemolytic anemia and pernicious anemia from other anemias which present with similar clinical and laboratory findings. In pernicious anemia, the intestinal absorption of Cobalt-60 labeled Vitamin B-12 can be measured by determining the percentage of radioactivity excreted in the urine and stool or by the percentage of radioactivity present in the liver area. The vagaries of stool and urine collection makes liver counting more practical for routine use. Homologous erythrocytes can be labeled with radioactive chromium 51 and reinjected. Measurement of the percent of radioactivity remaining on the eighth day gives information of clinical use in the diagnosis of the hemolytic state.

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A FAMILY REUNION: A STUDY OF HEREDITARY HEMORRHAGIC TELEANGIECTASIA

ROBERT M. BIRD,* JAMES F. HAMMARSTEN,** RICHARD A. MARSHALL,*** and R. R. ROBINSON****

New England Journal of Medicine 257:109-109, July 18, 1957

A large family in which 32 of 170 members were affected with hereditary hemorrhagic telangiectasia was studied. The mode of transmission was that of a simple autosomal dominant, and atavism did not occur. Genetic linkage between hereditary hemorrhagic telangiectasia and certain blood groups was sought but not found.

The typical lesion is a macule that resembles a crushed spider, about two to three mm. in diameter, cherry red in color, and not elevated. Great pressure is required to obscure these lesions, and at no time are definite pulsations observed. While the lesions did not pulsate, hemorrhage from the lesions was profuse and pulsatile. The most common sites of hemorrhage were nose, mouth, gastrointestinal tract and genitourinary tract. The lesions were observed in descending order of frequency on the face, lips, nares, tongue, ears, hands, feet and chest. The lesions were seldom found before the third decade and became increasingly frequent with age. Hemorrhage was more severe past 50 years of age and appeared to be more severe in males.

The family showed a remarkable adjustment to a life-threatening malady. Clannish attitudes had been fostered that robbed hemorrhage of its usual import. Independence and resourcefulness were given high value, and possible death from bleeding viewed objectively. Medical research into telangiectasia was encouraged by the group although there was no promise of immediate therapeutic gain.

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SYMPTOMATIC MAGNESIUM DEFICIENCY IN MAN

JAMES F. HAMMARSTEN* and WILLIAM O. SMITH**

New England Journal of Medicine 256:897-899, May 9, 1957

A patient is described in whom magnesium deficiency with confusion, stupor, tremers, and athetoid movements developed after profuse diarrhea and parenteral fluid therpay without added magnesium. The symptoms disappeared with magnesium sulfate therapy, reappeared when this therapy was discontinued and again subsided on reinstitution of the medication.

Magnesium deficiency has been described in chronic alcoholism with tremor, delirium, hallucinations, or convulsions. It has also been reported in patients with long-standing malignant osteolytic disease. The symptoms may be corrected by giving magnesium sulfate one or two gms. intramuscularly every four to six hours.

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HYPERTROPHIC OSTEOARTHROPY AND CHRONIC ULCERATIVE COLITIS

W. L. HONSKA, Jr.,* HENRY STRENGE,** and JAMES F. HAMMARSTEN***

Gastroenterology 33:489-492, September, 1957

Clubbing of the fingers and the toes has been mentioned as occurring with many gastrointestinal disorders which are accompanied by chronic diarrhea. However, hypertrophic osteoarthropathy with the characteristic periosteal proliferation is infrequently seen in association with ulcerative colitis. The authors report a 10-year-old male who had anemia, diarrhea, weight loss, and clubbing of the fingers and toes for four years. He had been repeatedly examined for congenital heart disease and this diagnosis never confirmed. The x-ray film of the radius and ulna showed the periosteal new bone formation which is the sin qua non of hypertrophic osteoarthropathy. The barium enema demonstrated ulcerative colitis. The occurrence of hypertrophic osteoarthropathy secondary to diseases not involving the lungs emphasizes that the syndrome is not always of pulmonary origin. Children with this disorder should be examined for diseases of the gastrointestinal tract.

*Medical Resident, VA and University Hospitals.

**Professor of Pediatrics, University of Oklahoma (presently in private practice in Boulder, Colorado).

***Associate Professor of Medicine, University of Oklahoma School of Medicine, and Chief, Medical Service, VA Hospital.

RESEARCH FOUNDATION

Six major areas of research are represented in the Foundation, ranging from microbiological studies in bacteria to elaborate metabolic balance experiments on patients in the Research Hospital. The work of three sections will be presented in this issue.

* * *

The Biochemistry Section, headed by Doctor Ranwel Caputto, is devoting considerable attention to the relatively little known metabolic functions of Vitamin E. Ascorbic acid synthesis, protein synthesis, and the composition of muscle are being studied in Vitamin E deficient animals. Doctor Caputto's group has recently announced the discovery in muscle of three compounds, heretofore unknown in this tissue. The basic work of this section in muscle biochemistry excites particularly the attention of those interested in the function, growth and wasting of muscle. Future bulletins will describe this work, now in preliminary stages, more fully. For papers published by this Section, see References 1, 2, 3.

The Cardiovascular Section, headed by Doctor Robert H. Furman, is approaching the problem of coronary artery disease through a study of blood fats (lipids). Struck by the tenfold higher incidence of coronary occlusions in men, and the presence of cholesterol in the coronary artery lesion, Doctor Furman's group has made an extensive study of blood fat patterns in men, women, and patients with various endocrine and blood lipid disorders. The blood fats studied, phospholipids and cholesterol, occur largely in combination with proteins (lipoproteins).

They are determined both chemically and after separation in the ultracentrifuge (high speed centrifugation). The latter method permits separation of lipoproteins of varying density, using the same principal by which cream is separated from milk. Mixing the blood fats in solvents of gradually increasing density, lipoproteins of increasing density, can be made to float to the surface.

Analysis of the lipoproteins, formerly done by a tedious optical technique, is now performed by an ingenious and more reliable method, devised recently and described by Doctor Furman and his associates.[4] The serum samples are spun in plastic tubes, and, immediately on removal from the centrifuge, are frozen in dry ice and acetone. The lipoprotein fraction, layered at the top of the tube, can then be separated from the solution below, simply and accurately, by sawing through the tube beneath the fatty layer. The method represents a significant technical advance for studies in this field.

Two of the lipoprotein fractions examined have proved to be peculiarly related to the endocrine status of the subject, the high density lipoproteins (referred to as "alpha") and lower density lipoproteins (referred to as "beta"). These relationships are summarized succinctly in a paper presented recently at the American College of Physicians Meeting in Boston by Doctors Furman and R. Palmer Howard:[5]

> "It is clear from these studies that the serum high density (alpha) lipoproteins and the ratio of high density to lower density (alpha/beta) lipoproteins are sensitive indicators of change in gonadal hormone relationships. Increase in relative estrogenicity, whether by administering estrogen or withdrawing androgen, is always associated with increase in high density lipoprotein concentrations and high density/low density lipoprotein ratios. Increase in relative androgenicity has the opposite effect. The lower density (beta) lipoprotein response is more variable but usually opposite to that of the high density fraction.
>
> "Changes in chemically determined cholesterol and phospholipid levels appear to be determined by the nature of the changes in the major lipoprotein fractions under these conditions. On the other hand, absence of change in serum cholesterol or phospholipid levels does not preclude the possibility of major alteration of the lipoprotein spectrum.
>
> "Whether these serum lipoprotein-hormone relationships are casually related to the marked sex difference in the incidence of coronary artery disease remains to be determined by further study. There is a good agreement among workers in this field that the concentration and physical status of the serum lipids have some etiologic significance in this disease. We feel, therefore, that

further efforts in the direction of the studies described are warranted."

A detailed description of this work appears in the January, 1958, issue of the *American Journal of Medicine*.[6]

Prednisone and cortisone produce changes in the lipoprotein pattern similar to those produced by estrogens. These findings and others, determined by the freeze-cutting technique, were presented to the Central Society for Clinical Research by Doctor Furman and colleagues in November, 1957.[4]

Blood lipid and lipoprotein changes in treated hypothyroid and hyperthyroid patients, and in euthyroid subjects receiving triiodothyroacetic acid (Triac) were described by Doctor Furman and his associates in a paper presented to the American Society for the Study of Arteriosclerosis, in Chicago, in November, 1957.[7] For other recent papers from this section, see References 8, 9, 10.

The Cancer Section, headed by Doctor Leonard P. Eliel, is engaged in endocrine and chemotherapeutic studies of breast carcinoma and leukemia. The mechanism of action and relative therapeutic efficacy of steroids, antifolic agents, and of hypophysectomy in breast carcinoma have been under investigation for the past three years. The effectiveness of various combinations and sequences of agents in the therapy of acute leukemia of children has also been the subject of extensive study.

Metabolic disturbances in patients with bone metastases are frequent, particularly hypercalcemia and hypercalciuria. Recently, abnormally low serum magnesium levels were encountered by the Cancer Section in a small group of patients. This study was presented to the American Society for Cancer Research in Chicago, April, 1957, and is summarized in the following abstract:[11]

The **Pathogenesis** and **Management** of **Metabolic Disturbances** in **Osteolytic Bone Disease**, Leonard P. Eliel, M.D., William O. Smith, M.D., and Agustin S. Alvarez, M.D.

"Metabolic studies in four patients with malignant osteolytic disease have revealed, in addition to hypercalciuria, hypercalcemia, and impaired renal function; hypomagnesemia and renal losses of magnesium in excess of theoretical contributions from destroyed bone. The magnesium losses varied directly with urine calcium. Convulsions were noted in three patients in association with periods of magnesium loss and hypomagnesemia.

"Intravenous administration of a chelating agent resulted in increased urine calcium, but no increment in magnesium excretion resulted.

"Intravenous calcium loading, sufficient to produce hypercalcemia, in two patients without osteolytic disease and with normal renal function, resulted likewise in lowered serum magnesium levels, but striking reductions in urine magnesium occurred, suggesting intracellular ingress, rather than loss, of magnesium.

"Effective reductions of urine calcium and comparable increases in fecal calcium, were observed promptly on institution of protein-free diets in patients with osteolytic bone disease. This measure may be as effective as oral chelating agents and tends to minimize renal damage until specific chemotherapeautic or endocrine measures to curb osteolysis becomes effective.

"It is concluded that 1) magnesium deficit (with convulsive tendencies) may accompany osteolytic bone disease and appears to be conditioned by urinary calcium ion concentration and renal damage, and 2) protein-free diets are an effective adjunct in the management of hypercalciuria and hypercalcemia."

The chemical similarity and associated clinical disturbances of calcium and magnesium, led Doctor Eliel and his group to investigate the possibility that parathyroid function influences the metabolism of magnesium. This work is summarized in the the following abstract,[12] and with the results of the above study, was presented by Doctors Eliel and Smith, at the first Oklahoma Colloquia on Advances in Medicine, February 8, 1958.[13]

Some Endocrine, Renal and Nutritional Factors Governing Magnesium Metabolism, Leonard P. Eliel, Wm. O. Smith, Carl Thomsen, and Josephine Hawrylko.

"The effects on magnesium metabolism of parathormone, acetazolamide (Diamox), calcium and phosphate loading, protein deprivation, and Vitamin D have been studied in man, using the metabolic balance technique.

"Two of four patients with hypoparathyroidism exhibited hypomagnesemia. The administration

of parathormone resulted in: 1) disappearance of hypomagnesemia and hypocalcemia, and 2) parallel changes in urine magnesium and calcium, i.e., an initial drop followed by rises to values exceeding control levels. Vitamin D likewise produced parallel urine changes in a hypoparathyroid patient with normal serum magnesium.

"Acetazolamind resulted in disappearance of tetany and prompt phosphorous diuresis, but in only minor changes in serum and urine calcium and magnesium. Severe tetany appeared soon afterwards in association with a marked drop in serum magnesium.

"Intravenous calcium and phosphate loads were followed by falls in serum magnesium attributed to intracellular migration of this ion.

"Intestinal absorption of magnesium was decreased by dietary protein deprivation.

"These studies suggest that: 1) the blood level and renal excretion of magnesium are governed directly or indirectly by parathyroid activity, 2) hypomagnesemia may facilitate the appearance of hypoparathyroid tetany, 3) the serum magnesium concentration may share in the same physical chemical relationships governing calcium and phosphorus concentrations, and 4) intestinal absorption of magnesium, like calcium, is affected by protein intake."

REFERENCES

1. Incorporation of P^{32} in the Muscle by Normal and Thyrotoxic Resting Rats. Philip C. Johnson, Audrey F. Posey, Daniel R. Patrick and Ranwel Caputto. Scheduled for January issue, American Journal of Physiology.

2. Creatinine-Phosphate Utilization by Muscle Extracts of Rabbits on Vitamin E Deficient Diets. Mary Carpenter, Ranwel Caputto and Paul McCay. Proc. Soc. Exp. Biol. Med., in press.

3. The Acid-Soluble Nucleotides of Muscle of Normal, Choline-Deficient and Vitamin E Deficient Rabbits. Henry Mosley, Paul McCay, Joe Pinto and Ranwel Caputto. Proc. 13th Southwest Regional Meetings Am. Chem. Soc. 1957, Page 39.

4. Gonadal and Adrenal Steroid-Induced Changes in the Cholesterol and Phospholipid Content of Serum Lipoprotein Fractions Isolated by Simultaneous Centrifugation of Four Solvent Densities Followed by Quick Freezing and Tube Sawing. Furman, R. H., Howard, R. P., and Norcia, L. N.—J. Lab. & Clin. Med. 50:816, 1957.

5. The Influence of Gonadal Hormones on Serum Lipids and Lipoproteins. Studies in Normal and Hypogonadal Subjects. Furman, R. H., and Howard, R. P. Ann. Int. Med. 47:969, 1957.

6. Influence of Androgens, Estrogens and Related Steroids on Serum Lipids and Lipoproteins: Observations in Hypogonadal and Normal Human Subjects. Furman, R. H., and Howard, R. P., Am. J. Med., January, 1958, page 80.

7. Serum Lipid and Lipoprotein Response to Therapy of Abnormal Thyroid Function and to Triiodothyroacetic Acid (Triac) Administration of Euthyroid Subjects. Furman, R. H., Howard, R. P., and Norcia, L. N. Circulation 16:489, 1957.

8. Correlations Between High Density—$S_{1.21}$ 0-20 ("alpha") Serum Lipoprotein Level, Serum Lipid Phosphorus Concentration and the Degree of Esterification of Serum Cholesterol. Furman, R. H. Circulation 16:507, 1957.

9. Influence of Testosterone, Methyltestosterone, and dl-Ethionine on Canine Liver Lipids and Lipoproteins. Furman R. H., Norcia, L. N., Robinson, C. W., and Gonzalez, I. E. Am. J. Physiol. Dec. 1957.

10. The Influence of Autoxidation on the Chemical Assay of Cholesterol. Norcia, L. N., Journal of Am. Oil Chemists' Soc., January, 1958, page 25.

11. The Pathogenesis and Management of Metabolic Disturbances in Osteolytic Bone Disease. Eliel, Leonard P., Smith, William O., and Alvarez, Agustin S. Proc. Amer. Assoc. Cancer Research, 2, 198, 1957.

12. Some Endocrine, Renal and Nutritional Factors Governing Magnesium Metabolism. Eliel, Leonard P., Smith, William O., Thomsen, Carl and Hawrylko, Josephine. Amer. J. of Med. 1958 (in press).

13. Magnesium-Calcium Interrelationships. Eliel, Leonard P., Smith, William O. (To be published in Proceedings of Colloquium.)

Reprints will be sent promptly on request. Send requests to 825 N.E. 13th Street, Oklahoma City 4, Oklahoma.

PERSONAL ITEMS

Doctor Ranwel Caputto became head of the Biochemistry Section, July 1, 1957, replacing Doctor C. D. Kochakian who left to become Professor of Physiology at the University of Alabama.

Doctor Raoul Trucco became Biochemist and Associate Head of the Psychosomatic and Neuromuscular Section. July 1, 1957, replacing Doctor Caputto (see above).

Doctor Max Huffman left the Foundation December 1, 1957, to become Director of the Lasdon Foundation Laboratories, Colorado Springs, Colorado.

Doctor Robert H. Furman was named Associate Director of Research of the Oklahoma Medical Research Foundation on January 1, 1958. He continues as Head of the Cardiovascular Section.

PHYSICIANS SUPPORT FOUNDATION

Oklahoma Physicians, 660 of them, had pledged $150,655 to the support of the Foundation over the next three years, as of January 24, 1958. This most gratifying response and expression of interest is a source of great encouragement to the entire staff.

OTHER SECTIONS TO REPORT

In subsequent issues the activities and work of the Dental, Endocrinology, Psychosomatic and an affiliated section (Behavorial Science) will be described.

Medical News

Medicare ID Card Issued

Effective immediately, dependents under the "Medicare" program will be required to present a Uniformed Services Identification and Privilege Card (DD Form 1173) before receiving medical care. The card number should appear in item number 6 of the claim form for claims reflecting a period of care beginning subsequent to January 1, 1958. There are two exceptions to the mandatory use of the card for identification purposes: Children under ten years old will not have individual cards and cards will not be required in emergency situations.

Medicare ID Card

In addition to item 6, the expiration date, which is indicated on the card, must be shown in item 7 of the claim form. If the ID card is not available, reasons must be stated in the spaces provided for items 6 and 7 or an explanatory note should be attached to the form.

The issuing branch of service is made evident by the letters preceding the card number. For example, Army issued cards have a prefix of two alpha characters, Navy cards are prefixed by the letter N, Public Health by PH, Coast Guard by CG and Air Force by AF. Such designation does not necessarily reflect the sponsor's branch of service. Interservice arrangements have been made whereby one service may issue cards to dependents of another branch, depending upon the residence of the dependent and the location of the nearest military issuing facility.

Fifth International Congress of Internal Medicine to Meet In Philadelphia

The world's largest international gathering of scientists and clinicians concerned with internal medicine will take place in Philadelphia on April 24-26 at the Fifth International Congress of Internal Medicine. In issuing the program of the meeting, T. Grier Miller, M.D., Philadelphia, the Congress President, called attention to the fact that in addition to American's leading internists, 81 foreign speakers representing 27 other nations would participate in the Congress' scientific program.

Among the 81 foreign speakers will be leading physicians from the Soviet Union, Czechoslovakia, Hungary, Rumania, and Poland. In announcing their participation, Doctor Miller said, "We are particularly pleased at the acceptance by these physicians of our invitation to join us at this International Congress. Their participation emphasizes that medical science knows no geographical or political barriers. It also represents immediate voluntary and professional implementation of President Eisenhower's invitation given in his recent State of the Union Message to the Soviet Union to join with us in cooperative medical research for the betterment of the health of mankind throughout the world."

Information and registration forms for the Congress can be obtained by writing Mr. E. R. Loveland, Secretary-General, Fifth International Congress of Internal Medicine, 4200 Pine Street, Philadelphia 4, Pennsylvania.

New Mexico Doctors Take Stand On Welfare Problem

The New Mexico Medical Society, with a membership of 509 physicians, has been in the middle of a complex and controversial political storm during the last few weeks over the handling of the state's welfare program.

State President Samuel R. Ziegler reports that after long and heated debate and much publicity, the welfare department accepted the society's offer to render free medical care to welfare patients rather than accept a further reduction in payments from the welfare fund. The schedule originally accepted by physicians represented roughly one-fourth of a doctor's usual fee.

The doctors' offer came after the state's department of public welfare claimed that because of a gradual increase in the number of patients the medical pool fund, established in 1952 with state and federal money, was running dry. The fund is used to provide medical surgical, hospital, dental, and nursing care and drugs for welfare patients.

The department of public welfare wanted the doctors to reduce their fees. The doctors said "no" to that plan and, instead, offered to give free medical care. The welfare department agreed to accept the offer which is to be effective from January 31 to June 30 at which time the physicians will meet with welfare department officials and review the experience. Doctors hope that meanwhile the department will institute and enforce better screening of applicants in the department's 31 county offices.

Russians Invited to Aero Medical Meeting

Russian medical scientists have received a special invitation to attend and participate in the Aero Medical Association's meeting scheduled for March 24-26 in Washington. The meetings attract many foreign scientists, especially from Europe. The invitation to the Russians, sent to the Russian embassy by Ashton Graybiel, M.D., of Pensacola, urges them "to present papers on their current work in space medicine."

Southwestern Surgical Congress To Meet in Houston

The Tenth Annual Meeting of the Southwestern Surgical Congress will be held March 31, April 1-2, 1958, at the Shamrock Hilton Hotel, Houston, Texas.

Four guest speakers will be featured on the program: Ralph F. Bowers, M.D., Chief of Surgical Service, Veterans Administration, Kennedy General Hospital, Memphis, Tennessee; Oscar V. Batson, M.D., ScD. (Hon) Professor of Anatomy, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania; George Crile, Jr., M.D., Division of General Surgery, Cleveland Clinic, Cleveland, Ohio and James F. Nolan, M.D., Los Angeles, California, Assistant Clinical Professor of Obstetrics and Gynecology, University of Southern California School of Medicine, Los Angeles, California.

The program for the three-day meeting will consist of 20 widely diversified scientific papers, panel discussions, roundtable luncheons, and television presentation of "Cancer Surgery" and "Heart and Vascular Surgery."

Topics for the panel discussions are: "Pitfalls In Gallbladder Surgery," "Factors Influencing Results of Cancer Surgery," and "Endometriosis." Questions regarding these topics may be submitted to the Central Office in advance. Address: Southwestern Surgical Congress, 813 Medical Arts Building, Oklahoma City 2, Oklahoma.

"Physicians and Social Security Inclusions"

Marjorie Shearon, who has written a Washington newsletter for many years, devoted her last issue to pro-and-con arguments regarding the inclusion of physicians under social security.

Physicians who are interested in reading Mrs. Shearon's informative and timely article can obtain a copy by writing to her at 9127 Jones Mill Road, Chevy Chase 15, Maryland. Single copies are 15 cents.

Junior Chamber Bestows Honors on Five Physicians

For the 20th consecutive year, the United States Junior Chamber of Commerce named 10 "outstanding young men of 1957" and five of them were physicians. The five doctors were:

Thomas G. Baffes, M.D., Skokie, Illinois surgeon, who developed the first surgical correction for transposing the great vessels of the heart.

Richard A. De Wall, M.D., Minneapolis, Minnesota surgeon, who developed a simplified mechanical heart-lung machine used in heart surgery.

Keith E. Jensen, M.D., Montgomery, Alabama microbiologist, who has conducted extensive studies of influenza viruses, including the recent Asian strain.

Robert E. L. Nesbitt, Jr., M.D., Albany, New York who has done research in the cause and prevention of infant death in association with the birth process. At 33, Doctor Nesbitt is the youngest chairman of a department of obstetrics and gynecology (Albany Medical College of Union University) in the nation's eighty-three medical schools.

David G. Simons, M.D., physician-scientist at Holloman Air Force Base, New Mexico, who broke records for free-balloon flight, reaching 102,000 feet last August.

Income of Russian Doctors

Senator Thye (R., Minn.), quoting reports received by him from an engineering professor who visited Russia, says in the Congressional Record that the medical doctor or attorney in that country can expect to earn the equivalent of between $325 and $450 per month, while a university professor will be getting $1,500 to $2,750 plus such extra benefits as autos at no cost. Experienced engineers are paid about what doctors are, high school teachers $300 to $325, common labor about $125. Ordinary living costs in Russia are reflected in the price of shoes: ordinary, $25; good quality, $75 to $100.

Court Upholds Disciplinary Act

Ralph Neill, executive secretary of the Washington State Medical Society, reports that the Supreme Court of Washington has affirmed the action of the disciplinary board operating under the Washington medical disciplinary act.

The board had suspended the license of a physician who was convicted of violating the internal revenue code of filing a false and fraudulent income tax return. This is believed to be the first case to have come before the Supreme Court of Washington under the medical disciplinary act which was adopted in 1955.

As soon as the opinion of the case becomes available, the A.M.A. law department will abstract it for publication in the *Journal* of the A.M.A.

Record Established in Handicapped Placements in Oklahoma in 1957

There were 11,981 handicapped workers placed on the job in 1957 by the Oklahoma State Employment Service. This was 2,916 above the 1956 placements—monthly increases in 1957 varied from a +47 to a high of +471. Oklahoma was *tenth* in the nation in handicapped placements in January, 1957, but presently ranks *fifth*—New York, Texas, Ohio and Pennsylvania are the only states leading Oklahoma in number of handicapped placed.

Fifth Bahamas Medical Conference To Be Held April 1-12

The Fifth Bahamas Medical Conference will be held at the Dolphin Hotel in Nassau, April 1-12, 1958, according to B. L. Frank, M.D., chairman of the Conference. Several outstanding speakers from various fields of medicine will be featured on the program.

Each physician registering at the conference will receive a certificate of attendance. Registrants and their families will enjoy reduced rates at the Dolphin; $14 per person per day including breakfast and dinner. All reservations should be made by writing directly to The Manager, The Dolphin Hotel, Nassau, Bahamas.

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER

Postgraduate Instruction*

PEDIATRIC SURGERY, RADIOLOGY, PATHOLOGY—March 14 and 15

Fourth Annual Combined Symposium
Sponsored by Oklahoma Association of Pathologists, Oklahoma Association of Radiologists, Oklahoma Chapter, American College of Surgeons

Guest Lecturers:

Robert E. Gross, M.D., Surgeon, Boston, Mass.
William L. Riger, M.D., Surgeon, Chicago, Ill.
Orvar Swenson, M.D., Surgeon, Boston, Mass.
John W. Hope, M.D., Radiologist, Philadelphia, Pa.
Also a prominent anesthesiologist and pathologist will be obtained for this program.

BASIC ELECTROCARDIOGRAPHY—March 31-April 5

This course consists of informal lecture presentations which assume no formal acquaintance with the subject. Laboratory exercises are carried out by the participants with individual help from the instructors. All working materials are furnished. Participants are expected to attend all lectures and laboratory periods and remains the entire time scheduled.

TRAUMA—April 11 and 12

Sponsored by the Regional Committee on Trauma of the American College of Surgeons

OKLAHOMA ASSOCIATION OF HOUSE STAFF PHYSICIANS—May 23

Two guest lecturers and presentation of original papers by members of the various House Staffs will highlight this program. Participating hospitals are: Hillcrest Medical Center, Tulsa; St. John's, Tulsa; McBride, Mercy, St. Anthony, University, Veterans Administration and Wesley, Oklahoma City.

*The above courses will be held at the University of Oklahoma School of Medicine. For further information write to the Office of Postgraduate Instruction, 801 NE 13th St., Oklahoma City, Oklahoma.

Postgraduate Division
UNIVERSITY OF OKLAHOMA MEDICAL CENTER
SHORT COURSE SERIES

Mar. 12—Medicine—Pathogensis and Treatment o Anemia.

April 9—Anesthesiology—Anesthesia for the Part-Time Anesthetist.

May 14—Pediatrics—Pediatric Allergy.

June 11—Surgery—Surgical Diagnosis and Problem Clinic.

The courses are designed so physicians may attend four hours of formal instruction in the above fields while spending only one-half day from their office. This series is approved for credit by the Oklahoma Academy of General Practice.

Time 3:30 to 8:30 p.m. the second Wednesday of each month.

Place: Room 120, Medical School Building.

Registration: $3.50 per session; $25 for the entire series includes dinner, Hospital Cafeteria. Mail registration to office of Postgraduate Instruction.

THE HEART PATIENT COURSE

April 21-May 2 Oklahoma Medical Center
Oklahoma City, Oklahoma

The Heart Patient Course, sponsored by the Oklahoma State Heart Association, Oklahoma State Department of Health, University of Oklahoma Medical Center, and the Oklahoma University Extension Division will be held April 21-May 2 at the Medical Center.

Two hours credit from the University of Oklahoma will be given to each enrollee who successfully completes the course which is particularly designed for physicians, nurses, dieticians, and social workers.

Scholarships for the $25 tuition fee are available to Oklahoma residents. For further information and application blank, write the Oklahoma State Heart Association, 825 Northeast 13th, Oklahoma City 4, Oklahoma.

O.S.M.A. ANNUAL MEETING

May 5, 6, 7

The 52nd Annual Meeting of the O.S.M.A. will be held in the Zebra Room of the Municipal Auditorium, May 5, 6, 7. Eleven nationally known physicians will be guest speakers.

Postgraduate Conference
University of Colorado Medical Center
Denver, Colorado
March 13-15, 1958

EDEMA—ITS PATHOGENESIS AND MANAGEMENT

This three-day postgraduate conference will be devoted to the basic considerations and clinical applications of kidney function, edema, and diuresis. It is designed to present in a comprehensive manner the problems of pathogenesis and management of edema as variously encountered in clinical medicine. Special emphasis will be placed on treatment.

A detailed program and further information may be obtained by writing to: The Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado.

HILLCREST MEDICAL CENTER
1653 East 12th St., Tulsa, Okla.

Lectures in Basic Science Given by Faculty Members of the University of Oklahoma School of Medicine.

Feb. 25—Common Metabolic Pathways, A. T. Bever, Lecturer.

Mar. 11—Anticoagulants, E. G. Larsen, Lecturer.
Mar. 25—Estrogens and androgens, A. T. Bever, Lecturer.

April 9—Adrenal Steroids; Aldosteronism, R. W. Payne, Lecturer.

April 30—Thyroid: Recent Advances in Diagnosis and Treatment, R. W. Payne, Lecturer.

May 13—Pulmonary Function, M. T. Lategola, Lecturer.

May 27—Basic GI Phisiology, J. W. H. Smith, Lecturer.

June 10—Physiological Basis of Liver Function Tests, E. G. Larsen, Lecturer.

June 24—Alimentary Reserve: the malabsorption syndrome, J. W. H. Smith, Lecturer.

Tenth Annual Meeting
SOUTHWESTERN SURGICAL CONGRESS
March 31, April 1-2
Shamrock Hilton Hotel Houston, Texas

Guest speakers for this meeting will be: Ralph F. Bowers, M.D., Memphis, Tennessee; Oscar V. Batson, M.D., ScD. (Hon), Philadelphia, Pennsylvania; George Crile, Jr., M.D., Cleveland, Ohio; and James F. Nolan, M.D., Los Angeles, California.

Further information may be obtained by writing to the Central Office, Southwestern Surgical Congress, 813 Medical Arts Building, Oklahoma City 2, Oklahoma.

Have You Heard?

ALFRED R. SUGG, M.D., Ada, has been appointed a member of the Governor's Committee on Employment of the Handicapped. Object of the committee is to promote increased employment opportunities on a year-round basis for handicapped workers and to promote better understanding of problems of the handicapped.

HASKELL R. ANDERSON, M.D., recently terminated his practice in Watonga. Doctor Anderson announced that he planned to reenter Public Health Service work some time in the future.

The Journal of the A.M.A. published an article by A. R. STOUGH, M.D., McAlester, in its February 22 issue. Doctor Stough's article reported that no habituation to the tranquilizer meprobamate developed among sixty prison inmates given the drug during a recent study.

CHARLES A. CASHMAN, M.D., who has been in practice in Shidler and Okemah since 1953, moved to Hobart in March where he will be associated with J. WILLIAM FINCH, M.D., at the Hobart Clinic.

PORT JOHNSON, M.D. was guest speaker at the Pittsburg County Medical Society meeting in February. His subject was "Non-operative Management of Low Back Pain."

SOCIAL SECURITY SAYS: "A woman who becomes entitled to benefits based on her own earnings and also the wife's benefits on the earnings of her husband would receive no more than the larger of the two amounts. A child who becomes entitled to child's benefits based on earnings of both his father and mother would not receive both payments."

In Other Words: Double social security taxes paid by one family do not produce benefits for each member paying the taxes. A part of the taxes go to "charity."—*Prepared by the A.M.A.*

Organization News

"Menopause—Facts and Fables" Topic for Annual Meeting

John E. Hobbs, M.D., will be one of eleven outstanding guest speakers when the Oklahoma State Medical Association convenes at its fifty-second annual meeting in Oklahoma City, May 5. Doctor Hobbs will speak on the subject of "Endometriosis" on May 5 and will appear again on the program of May 6 when he discusses "Menopause—Facts and Fables." An authority in his field, he is an Associate Clinical Professor of Obstetrics and Gynecology at the Washington University School of Medicine, St. Louis, Missouri.

JOHN E. HOBBS, M.D.

Other out of state speakers will be: J. Arnold Bargen, M.D., gastroenterologist, Mayo Clinic and President of the Minnesota State Medical Association; Charles H. Brown, M.D., gastroenterologist, Cleveland Clinic; John H. Githens, M.D., pediatrician, University of Colorado; Alvin J. Ingram, M.D., orthopedist, Campbell Clinic, Memphis; Kenneth C. Johnston, M.D., endoscopist, Chicago; Charles W. Mayo, M.D., surgeon, Mayo Clinic; Robert D. Moreton, M.D. radiologist, Ft. Worth; Edgar J. Poth, M.D. surgeon, University of Texas; Edwin L Prien, M.D., urologist, Brookline, Massachusetts; and Louis A. Soloff, M.D., cardiologist, Temple University.

The meeting will be held in the Zebra Room, Municipal Auditorium, May 5, 6, and 7. Preceding the scientific sessions, the House of Delegates will meet on Sunday, May 4, with the first session convening at 10:30 a.m. in the Hall of Mirrors at the Auditorium.

Social events associated with the meeting will be a dinner dance and a Blue Shield-sponsored dinner. The dance, featuring the Charlie Spivak Orchestra, will be held Tuesday evening, May 6 in the Persian Room of the Skirvin Tower Hotel. Although final plans for the Blue Shield affair are not definite, it will be complimentary to all members of the Association and their wives.

Honorary and Life Memberships Now Classed As One

As a reminder to county society secretaries, O.S.M.A. President, John F. Burton, M.D., requested that the *Journal* summarize action taken by the House of Delegates at the 1957 Annual Meeting regarding Honorary and Life Membership classifications.

The Life membership classification was deleted from the By-Laws and Chapter 1, Section 3, Subsection (b) was amended to read as follows:

"Any physician, a member of this Association, who by reason of ill health or age shall retire from the active practice of medicine, or who has been engaged in the active practice of medicine fifty years, or more and whose service to humanity and his profession has been conducted with dignity and honor may be placed on the Honorary-Life Membership roll. Eligibility for such consideration is limited to those physicians who

have been members of this Association not less than five years immediately preceeding application and whose petition for such membership is initiated by a component society of this Association, presented for consideration to the Council, and approved by the Council at a meeting prior to the Annual Session.

"The approval of the House of Delegates, by a majority vote thereof at the annual session, shall be necessary to place such eligible members on the Honorary-Life Membership roll. Such members shall have all the privileges of active membership except holding office, and shall not be required to pay dues or assessments in this Association. Honorary-Life Members shall be considered the same as fully-paid members in computing the membership of the County Societies for the purpose of determining the number of Delegates that the County Societies shall be entitled to send to the House of Delegates as provided by these By-Laws."

These changes were prompted by general confusion between the significance of the two awards and it was felt that the consolidation motion would provide the necessary corrective measure. The appropriate applications were sent to county medical society secretaries. Additional copies may be obtained by writing the state office.

In a separate action, the House of Delegates provided for the recognition of outstanding accomplishments in the field of medicine by adding a subsection (b) to Chapter III, Section 6, the portion of the By-Laws pertaining to Memorials and Resolutions. The new subsection reads as follows:

"Accomplishments in the field of medicine may be recognized by an award of a certificate of accomplishment. Such award shall be initiated by a component society, approved by the Council prior to the Annual Session, and voted by the House of Delegates. To be eligible for such an award, a physician shall have been a member of the Association for not less than five years immediately preceding his recommendation therefor." Forms for the purpose of initiating such an award are being mailed to county society secretaries.

Doctors To Head West for A.M.A. Annual June Meeting

Between 12,000 and 15,000 physicians will journey westward in June in search of something far more valuable than gold. They'll be on a quest for the latest information on new medical techniques and discoveries at the American Medical Association's 107th Annual Meeting in San Francisco. The five days of June 23-27 will be filled with bright nuggets — including scientific exhibits, lectures, motion pictures, panel discussions, televised surgical procedures and commercial exhibits. Convenient center for the Scientific and Technical Exhibits, films, color TV and lectures will be the Civic Auditorium, the adjacent new Plaza Exhibit Hall and other surrounding buildings. Headquarters for the House of Delegates sessions will be the Sheraton-Palace Hotel.

Plans for an outstanding scientific lecture program are being completed by the Council on Scientific Assembly. Opening the general scientific program Monday afternoon, June 23, will be a symposium on the care of the severely injured patient. Tuesday morning a general meeting will feature another symposium on hazards associated with therapeutic agents. Formal scientific section meetings will run from Tuesday afternoon through Friday morning.

Special panel discussions and demonstrations are being planned throughout the meeting, including: perinatal problems; methods of resuscitation of infants; nutrition; physical examination of physicians, using electrocardiograms and chest x-rays; fresh tissue pathology, and treatment of fractures. The Section on Miscellaneous Topics also is planning sessions on allergy, prevention of traffic accidents, prevention of injury in sports, and medical professional liability. Other features will be a color television program of live operations and demonstrations from San Francisco Hospital and a varied motion picture program.

Registration officially opens at the new Plaza Exhibit Hall Monday, June 23, at 8:30 a.m. and closes Friday noon. Advance registrations will be accepted Sunday, June 22, from 12 noon to 4:00 p.m. The Scientific

and Technical Exhibits will be open to A.M.A. physician-members *only* on Tuesday and Wednesday mornings.

Two high school winners of A.M.A. scientific awards at the National Science Fair again will display their prize exhibits. In addition, the top winners of the intern-resident and medical student exhibit classifications at the Student A.M.A. convention this spring will be invited for the first time to exhibit at an A.M.A. meeting.

Plan now to attend this worthwhile medical meeting. Watch for further details in *The Journal of the A.M.A.*

American Academy of General Practice Meets in Tulsa

Over 200 physicians registered for the tenth annual meeting of the Oklahoma Chapter of the American Academy of General Practice which was held in Tulsa, February 3-4.

Guest speakers for the two-day meeting were: Justin J. Cordonnier, M.D., St. Louis, Missouri; Michael E. DeBakey, M.D., Houston, Texas; E. Richard Harrell, Jr., M.D., Ann Arbor, Michian; Paul C. Laybourne, M.D., Kansas City, Kansas; Benjamin H. Robbins, M.D., Nashville, Tennessee, and Richard H. Shepard, M.D., Baltimore, Maryland.

A banquet was held on Monday evening in the Topaz Room, Tulsa Hotel. Entertainment was furnished by the "Kenneth Anquoe Indian Troupe" and "The Tulsans," a male chorus, directed by Ronald Shirey. Guest speaker for the evening was Malcom E. Phelps, M.D., El Reno, President of the American Academy of General Practice.

Newly elected officers of the group are: President, Charles E. Wilbanks, M.D., Tulsa; President-Elect, Roger Reid, M.D., Ardmore; Vice-President, Robert T. Sturm, M.D., Oklahoma City; Secretary-Treasurer, V. M. Rutherford, M.D., Midwest City; Delegates, Marshall O. Hart, M.D., Tulsa and M. B. Glismann, M.D., Oklahoma City; and Alternates, Edward T. Cook, M.D., Anadarko and Mark D. Holcomb, M.D., Enid.

Physicians Reminded Of Birth, Death Laws

The Division of Statistics of the Oklahoma State Department of Health has asked that members of the association be reminded of certain requirements outlined in the Oklahoma Statutes pertaining to the reporting of births and deaths.

REGISTRATION OF BIRTHS

Oklahoma Statute 63 O. S. 1951 § 560.3

A. Within the time prescribed by the commissioner a certificate of every birth shall be filed with the registrar of the district in which the birth occurred by the physician, midwife, or other legally authorized person in attendance at the birth; or if not so attended, by one of the parents.

Resolution adopted in July 1948 pursuant to the above statute.

(1) Birth registration. Every live birth which shall occur in this state shall be filed with the local registrar of the district in which the birth occurred within 10 days after the date of birth.

REGISTRATION OF DEATHS AND STILLBIRTHS

Oklahoma Statute 63 O. S. 1951 § 560.4

A certificate of every death or stillbirth shall be filed with the local registrar of the district in which the death or stillbirth occurred within 3 days after the occurrence is known; or if the place of death or stillbirth is not known then with the local registrar of the district in which the body is found within 24 hours thereafter. In every instance a certificate shall be filed prior to interment or other disposition of the body.

B. (2) In preparing a certificate of death or stillbirth the person in charge of interment shall obtain and enter on the certificate the personal data required by the commissioner from the persons best qualified to supply them. He shall present the certificate of death or stillbirth to the physician last in attendance upon the deceased.

C. If the cause of death cannot be determined within three days, the certification of its cause may be filed after the prescribed period, but the attending physician shall give the local registrar of the district in which death occurred, written notice of the reason for the delay, in order that a permit for the disposition of the body may be issued.

OFFENSES

Oklahoma Statute 63 O. S. 1951 § 560.13

C. Except where a different penalty is provided in this section, any person who violates any of the provisions of this Act or neglects or refuses to perform any of the duties imposed upon him by this Act, shall be fined not more than One Hrundred Dollars ($100.00).

OSMA Representatives Attend AMA Meeting in Dallas

Representatives of the Oklahoma State Medical Association were present at a meeting of the A.M.A.'s Committee on Prepayment Medical and Hospital Service which was held February 28 in the Adolphus Hotel, Dallas.

President John F. Burton, M.D., President-Elect E. C. Mohler, M.D., Past-President H. M. McClure, M.D., and Executive Secretary Don Blair were in attendance. Doctor Mohler appeared on a panel concerned with "problem areas in voluntary health insurance."

In addition to Oklahoma, representatives were present from Alabama, Arkansas, Kansas, Louisiana, Mississippi, New Mexico, Texas, and the Dallas County Medical Society. The regional meeting with the Committee is under the auspices of the A.M.A.'s Council on Medical Service, of which the Committee is a sub-division.

Other discussions were centered around the profession's role in voluntary health insurance, degree of control to be exercised, summary of the background and growth of such plans, and future attitudes toward such programs.

Upper Respiratory Disease Rate Drops

Following several weeks of slight increases, the rate for upper respiratory disease (resulting in a day or more in bed) is showing a decrease, according to the National Health Survey. Revised figures show 3,683,000 new cases for the week ending January 11, but a drop to 2,274,000 for the next week. Similarly, the average number of persons in bed because of these diseases dropped by 20 per cent, to 1,644,000. Both estimates for new cases and average daily number of bed cases are now at the lowest levels since September, according to the Survey. Survey statistics lag three or four weeks behind because they are compiled from door-to-door questioning of a cross-section of the population.

Revised Constitution Drafted

The Constitution and By-Laws Committee of the Oklahoma State Medical Association, at the direction of the Council, has prepared a revised constitution and by-laws which will be presented to the House of Delegates at the annual meeting in Oklahoma City on May 4.

Members of the committee are Chester McHenry, M.D. (Chairman), William T. Gill, M.D. and Clinton Gallaher. M.D.

Copies of the proposed changes to the by-laws will be mailed to county society presidents and secretaries as well as to all delegates to the O.S.M.A., prior to the annual meeting. In accordance with the present constitution, the amended constitution is printed below for the information of all members of the association.

CONSTITUTION

ARTICLE I.

The name of this organization is the OKLAHOMA STATE MEDICAL ASSOCIATION, INCORPORATED.

ARTICLE II.

Purpose of the Association

This Association is formed to promote the science and art of medicine.

ARTICLE III.

Component Societies

The Association shall charter component county or district societies.

ARTICLE IV.

Membership

The members of the component societies shall be the members of the Association. Membership in the Association is a privilege and not a right.

ARTICLE V.

House of Delegates

Section 1. The House of Delegates shall be the legislative body of the Association. It shall be composed of (1) Delegates elected by the component societies, and (2) the general officers of the Association.

Section 2. It shall meet at least once at each annual meeting. A special meeting may be called by the president, the council, or by petition to the speaker from not less than thirty delegates. Only such business as is specified in the call shall be transacted at a special meeting.

Section 3. It shall apportion the State into suitable councilor districts.

ARTICLE VI.

The Council

Section 1. The Council shall consist of two councilors from each councilor district, and the other general officers of the Association.

Section 2. The Council shall be the executive and the judicial branch of the Association.

Section 3. The Council shall meet at least once during the annual meeting, and on call by the president, or by petition to the president from one-third or more of its members.

ARTICLE VII.

Meetings

The Association shall hold an annual meeting at a place designated by the House of Delegates. For good and sufficient reasons the Annual Meeting place may be changed by a three-fourths vote of the Council.

ARTICLE VIII.

General Officers

Section 1. The general officers of the Association shall be the President, President-Elect, Vice-President, Secretary-Treasurer, Speaker and Vice-Speaker of the House of Delegates, the Councilors, the Delegates and Alternate Delegates to the American Medical Association, the Editor of the Journal, and the most recent two past presidents of the Association.

Section 2. All general officers shall be elected by the House of Delegates at its Annual Meeting, except the Editor of the Journal, and may be removed from office for cause.

Section 3. The President-Elect and the Vice-President shall be elected for a term of one year. The Secretary-Treasurer, Speaker and Vice-Speaker of the House of Delegates shall be elected for a term of two years. The councilors shall be elected for a term of three years, the terms being so arranged that approximately one-third shall be elected each year. At the expiration of his term, the President-Elect shall become President for a term of one year.

Section 4. The general officers, except Delegates and Alternate to the American Medical Association who will not assume office until January 1, shall assume the duties of their offices at the close of the Annual Meeting at which they are elected, and shall serve until their successors have been elected and installed.

Section 5. Vacancies in any general office shall be filled by appointment by the President, with the approval of the Council, effective until the next annual meeting, at which time the House of Delegates shall elect a successor to complete the unexpired term, except in the case of the President or Speaker of the House of Delegates, or Delegates to the American Medical Association, where the Vice-President or Vice-Speaker shall automatically fill the vacancy.

ARTICLE IX.

Finances

The House of Delegates shall levy such dues and assessments as it considers proper for the conduct of the business of the Association. The council, at least annually, shall submit a budget for future operations of the Association to the House of Delegates, and the House of Delegates shall make such appropriation of funds as it considers proper.

ARTIVCLE X.

Referendum

The House of Delegates may submit any question to a vote of the members of the Association, provided that two-thirds of the Delegates present at the meeting vote in favor of such referendum. The House of Delegates shall designate a committee to conduct the referendum and publish the result.

ARTICLE XI.

Seal

The House of Delegates shall adopt an official seal for the Association.

ARTICLE XII.

Ethics

The principles of medical ethics of the American Medical Association currently in force shall be those of this Association, and shall govern the conduct of its members.

ARTICLE XIII.

Amendments

The House of Delegates may amend this constitution or any part thereof by the affirmative vote of two-thirds of the Delegates present at any annual meeting, provided that notice of the proposed amendment shall have been given to each component society at least 60 days before such meeting. No amendment shall become effective until the close of the annual meeting at which it is adopted.

ARTICLE XIV.

This Constitution supersedes and repeals all previous constitutions. Any By-law, Resolution, or enactment in conflict herewith is declared to be of no effect.

A.M.A. To Publish Tabloid Paper

The Board of Trustees, meeting in Chicago recently, authorized publication of a new. 16-page tabloid newspaper called *"The A.M.A. News."* The newspaper which is in its formative and planning stages is expected to be distributed every two weeks to approximately 200,000 physicians.

In this way, we will be able to bring to the attention of our members the multitude of projects and activities carried on by their association, as well as all non-scientific news of interest to the profession. Publishing a newspaper for members was selected as ameans of improving communications between the association and its members.

The *"A.M.A. News"* project was discussed with many people, inside and outside of medicine. In nearly every instance, it was felt that such a publication, built on a news format, was needed.

Physicians will be kept informed of what is going on in medicine in all 48 states through *"The A.M.A. News."*

The publication will carry advertising, and the editorial staff, yet to be selected, will work at the association's Chicago headquarters.

The first issue is expected to be ready for distribution at the A.M.A. annual convention in San Francisco, June 23-27.

M.D.'s, Lawyers Meet in Ardmore

The regular monthly meeting of the Carter-Love-Marshall Counties Medical Society was held as a joint meeting with members of the Carter County Bar Association.

The two groups assembled at the Tom Cooper Dairy Store for a dinner which was presided over by Tom Sparks II, M.D., President of the Medical Society. He was assisted by Frank Thomas, President of the Bar Association.

Following dinner, a general round table discussion was developed on Medico-legal problems, and methods of cooperation, in which almost everyone present participated. The members of each group felt they had greatly benefitted by the meeting and it was unanimously agreed to make it an annual affair to be held each February.

What's Your Hobby, Doctor?

The DOCTOR'S HOBBY SHOW has become one of the outstanding attractions at the OSMA ANNUAL MEETING. A project of the Woman's Auxiliary, the show offers physicians an excellent opportunity to display the products of their leisure time. If you have a hobby, don't keep it a secret . . . Show your colleagues what you can do . . . APPLY NOW!

Doctor's Hobby Show

O.S.M.A. Annual Meeting

Municipal Auditorium

Oklahoma City

MAY 5-6-7-, 1958

Application For Hobby Show Space

52nd ANNUAL MEETING

OKLAHOMA STATE MEDICAL ASSOCIATION

DESCRIBE EXHIBIT, including information as to size, shape and value (insurance is provided):

IMPORTANT: Deliver Exhibit to Zebra Room, Municipal Auditorium, by noon, May 4. Your Exhibit will be personally attended and insured at all times. It must be taken down by noon, May 7, when management responsibility ends.

MAIL THIS FORM TO:

Mrs. Wm. R. R. Loney, Chairman,
Doctor's Hobby Show
2440 East 26th Place
Tulsa 14, Oklahoma

APPLICATION FOR SPACE

SCIENTIFIC AND EDUCATIONAL EXHIBIT SECTION

52nd ANNUAL MEETING ★ OKLAHOMA STATE MEDICAL ASSOCIATION
MUNICIPAL AUDITORIUM ★ OKLAHOMA CITY ★ MAY 5, 6, 7, 1958

(Please type or print)

Name of organization or individual requesting space: _____

_____

_____

Please describe your exhibit (75 words maximum - to be used in official program:)

_____

_____

_____

_____

Amount of space required (indicate width in multiples of eight feet): _____ feet.
(All exhibit spaces will be 6 feet deep.)

If you have ever exhibited at an Annual Meeting, give most recent date: _____

Date of application: _____

Applications should be mailed to the Scientific and Educational Exhibits Committee, Oklahoma State Medical Association, Box 9696 Shartel Station, Oklahoma City.

Closing date for filing applications: March 15, 1958. Space is limited and not all applications can be accepted. There will be no charge for space, but each exhibitor is responsible for installation of own exhibit. The Oklahoma State Medical Association assumes no liability for the safety of exhibits. Exhibits will be installed on Sunday, May 4, 1958 and must be removed between 1:00 p.m. and 5:00 p.m. on Wednesday, May 7, 1958.

Doctor Kelso Speaks in Far East

Joseph W. Kelso, M.D., Oklahoma City is now on an extensive tour of the Far East where he will deliver scientific papers in several foreign countries. The initial invitation to speak at the Kumamoto Medical School in Japan prompted further speaking dates and he is now scheduled for three talks in the Philippine Islands and an additional two talks in Japan.

On March 17, he will speak to the Philippine Surgical Society in Manila. He will also speak at Olongapo-Zamboles on March 18 and the following day at Baguio, the summer capitol of the Philippines.

After leaving the Philippines, he and Mrs. Kelso will journey to Singapore, Bankok, Saigon, and Hong Kong before filling his speaking engagements in Japan.

He will be one of the two foreign speakers at the 19th Annual Meeting of the Japanese Obstetrical and Gynecological Society when they meet at Fukuoka, Japan. In addition to speaking on the "Surgical Treatment of Carcinoma of the Cervix," he will show his own movies on "Vaginal Hysterectomy" and "Radical Vulvaectomy and Bilateral Gland Resection."

At Kumamoto, he will talk on the "Management of Gynecological Urological Accidents" and the "Surgical Management of Carcinoma of the Cervix."

Following the Kumamoto presentations, he will travel to Tokyo where he will give the same addresses plus a paper entitled, "The Infertile Couple."

Upon return to the United States, he will speak before the American College of Obstetricians and Gynecologists on April 23 in Los Angeles. Enroute to Oklahoma City, he will stop in Pueblo, Colorado to deliver a paper.

AN INVITATION TO EXHIBIT

OSMA ANNUAL MEETING

Do you have an idea which you would like to present to other physicians in Oklahoma? An unusual case? A series of cases? A new device or technique? An organization problem or plan of action? A piece of experimental work?

If so, consider working it up as a SCIENTIFIC EXHIBIT to be shown at the annual meeting of the Oklahoma State Medical Association in May, 1958. Applications should be submitted on form reproduced on the opposite page.

Start now collecting your material and planning your exhibit. Since space is limited, you are encouraged to submit your application early. Your exhibit need not be large. It should be stimulating. Subjects which have special visual interest are particularly suitable.

MAY 5, 6, 7

ZEBRA ROOM **MUNICIPAL AUDITORIUM**

OKLAHOMA CITY

Deaths

Stephen W. Reynolds, M.D.

1891-1958

Stephen W. Reynolds, M.D., Drumright physician, died in his office February 3, 1958. Doctor Reynolds was born in St. Joseph, Missouri in 1891.

After graduating from the University of Oklahoma School of Medicine in 1914, he began his practice in Drumright.

Doctor Reynolds was a member of the Creek County Medical Society, the Oklahoma State Medical Association, and the American Medical Association.

Berthe Margolin Lee, M.D.

1894-1958

Berthe Margolin Lee, M.D., Tulsa anesthetist, died February 1, 1958 from injuries suffered when she was knocked down by her rolling automobile. Doctor Lee was the wife of J. K. Lee., M.D., also a Tulsa physician.

A native of Riga, Latvia, Doctor Lee, who was known professionally as Doctor Berthe Margolin, came to the United States in 1915. She graduated from the Fort Worth School of Medicine in 1917 and moved to Tulsa in 1918.

Doctor Lee was a member of the Tulsa County Medical Society, the Oklahoma State Medical Association, and the American Medical Association.

Udonna Clifton Boon, M.D.

1874-1958

Udonna Clifton Boon, M.D., 83-year-old pioneer Chickasha physician died in Round Lake, Illinois, February 8, 1958.

Doctor Boon was born in 1874 in Whitesburg, Georgia. He graduated from Tulane University School of Medicine in 1898.

In recognition of his fifty years of service to the medical profession, Doctor Boon was awarded a fifty-year pin by the Oklahoma State Medical Association.

Doctor Boon retired two years ago to make his home in Round Lake. He was a member of the Oklahoma State Medical Association and the American Medical Association.

Blair Points, M.D.

1885-1958

Blair Points, M.D., 73, who practiced medicine in Luther for 20 years, died February 10, 1958 in Oklahoma City. Doctor Points was the father of Thomas C. Points, M.D., Oklahoma City physician.

Doctor Points was a member of the first class to be graduated from the University of Oklahoma School of Medicine in 1911. He established the first full time county health unit in Oklahoma in Ottawa county in 1918.

Doctor Points was a member of the American Medical Association.

Continued from Page 114

SUMMARY AND REFERENCES FOR 'THE EFFECT OF DIGOXIN ON CARDIAC OUTPUT AND HEMODYNAMICS IN EXPERIMENTAL SUPRAVALVULAR MITRAL STENOSIS.*

Summary and Conclusions

1. Dogs in congestive heart failure from experimental mitral stenosis generally displayed a fall in cardiac output with a fall in pulmonary artery pressure.

2. Dogs, following phlebotomy, showed an essentially similar change.

3. Diuresis and naturesis was frequently observed with a fall in cardiac output and no appreciable change in right atrial pressure.

REFERENCES

1. Gray, F. D., Jr., Williams, M. H., and Gray, F. G. The circulatory and ventilatory changes in chronic pulmonary disease as affected by Lanatoside C. Am. Heart J. 44: 517, 1952.

2. Jaques, W. E. and Hyman, A. L. Experimental supravalvular mitral stenosis in the dog. A.M.A. Arch. Path. 64: 67, 1957.

3. McMichael, J. and Sharpey-Schafer, E. P. The action of intravenous digoxin in man. Quart. J. Med. 13: 123, 1944.

4. Dock, W. and Tainter, M. L. The circulatory changes after full therapeutic doses of digitalis with a critical discussion of views on cardiac output. J. Clin. Invest. 8: 467, 1930.

5. Ferrer, M. I., Harvey, R. M., Cathcart, R. T., Cournand, A. and Dickinson, W. R., Jr. Hemodynamic studies in rheumatic heart disease. Circ. 6: 688, 1952.

6. Baronofsky, I. D., and Haddy, J. H. Cardiopulmonary dynamics of mitral stenosis: A clinical and experimental study. Surg. 34: 347, 1953.

7. Hyman, A. L., Jaques, W. E., and Hyman, E. J. Observation of the direct effect of digoxin on renal excretion of sodium and water. Am. Heart J. 52: 592, 1956.

*This investigation was supported by Research Grant H-1838 and H-3235S from the National Heart Institute of the National Institutes of Health, Public Health Service.

**—Nembutal—Abbott Laboratories.

*—Digoxin—Burroughs Wellcome & Co.

(801 Northeast 13th Street, Oklahoma City, Oklahoma)

Continued from Page 117

SUMMARY AND REFERENCES FOR 'THE CONTROL OF A POST-NATAL EPIDEMIC OF BREAST ABSCESSES IN A GENERAL HOSPITAL.'

Summary

An epidemic of breast infections is presented. The methods of studying and combatting this epidemic are described, and the results are presented. Suggestions are made for the prevention and handling of similar situations.

REFERENCES

Caswell, H. T., and Burnett, W. E.: Chronic recurrent breast abscesses secondary to inversion of the nipple, Surg. Gynec. & Obst. 102:439, 1956.

Gibbard, G. F.: Sporodic and epidemic puerperal breast infections, Am. J. Obst. & Gynec. 65:1038, 1953.

Hesseltine, H. C., Freundlich, C. G., and Hite, K. E.: Acute puerperal mastitis, Am. J. Obst. & Gynec. 55:778, 1948

Hesseltine, H. C., and Priddle, H. D.: Treatment of acute puerperal mastitis, Am. J. Obst. & Gynec. 61:1370, 1951.

Hobbs, J. E.: A new method of treating breast abscesses, Surg. Gynec. & Obst. 54:839, 1932.

Newton, M., and Newton, N. R.: Breast abscesses, a result of lactation failure, Surg. Gynec. & Obst. 91:651, 1950.

Pyle, L. R.: Staphylococcus aureus hemolyticus puerperal mastitis and infection of the newborn, Am. J. Obst. & Gynec. 55:676, 1948.

Sawyer, C. D., and Walker, P. H.: A bacteriologic and clinical study of breast abscesses, Surg. Gynec. & Obst. 99:368, 1954.

(St. Johns Hospital, Tulsa, Oklahoma)

Continued from Page 119

REFERENCES FOR 'STOP RHEUMATIC FEVER.'

REFERENCES

1. Paul, John R.: The Epidemiology of Rheumatic Fever and Some of its Public Health Aspects—Metropolitan Life Insurance Company, pp. 163, 2nd Ed. 1943.

2. Quinn, Robert W.: Rheumatic Heart Disease and Crowding—Am. J. Public Health 38, 1071-1081, 1948.

3. Coburn, A. F. and Young, D. C.: The Epidemiology of Hemolytic Streptococcus During World War II in the United States Navy—Williams & Wilkins Co. pp 229 1949.

4. Wannamaker, Lewis W.: The Epidemiology of Streptococcal Infections. Chap. 12, pp. 157-175 in Streptococcal Infections edited by Maclyn McCarty, Columbia University Press, 1954.

5. Stetson, Chandler A.: The Relation of Antibody Response to Rheumatic Fever. Chapt. 15, pp. 208-218 in Streptococcal Infections edited by Maclyn McCarty, Columbia University Press, 1954.

6. Bernheimer, Alan W.: Streptolysins and Their Inhibitors. Chap. 2, pp. 19-38 in Streptococcal Infections edited by Maclyn McCarty, Columbia University Press, 1954.

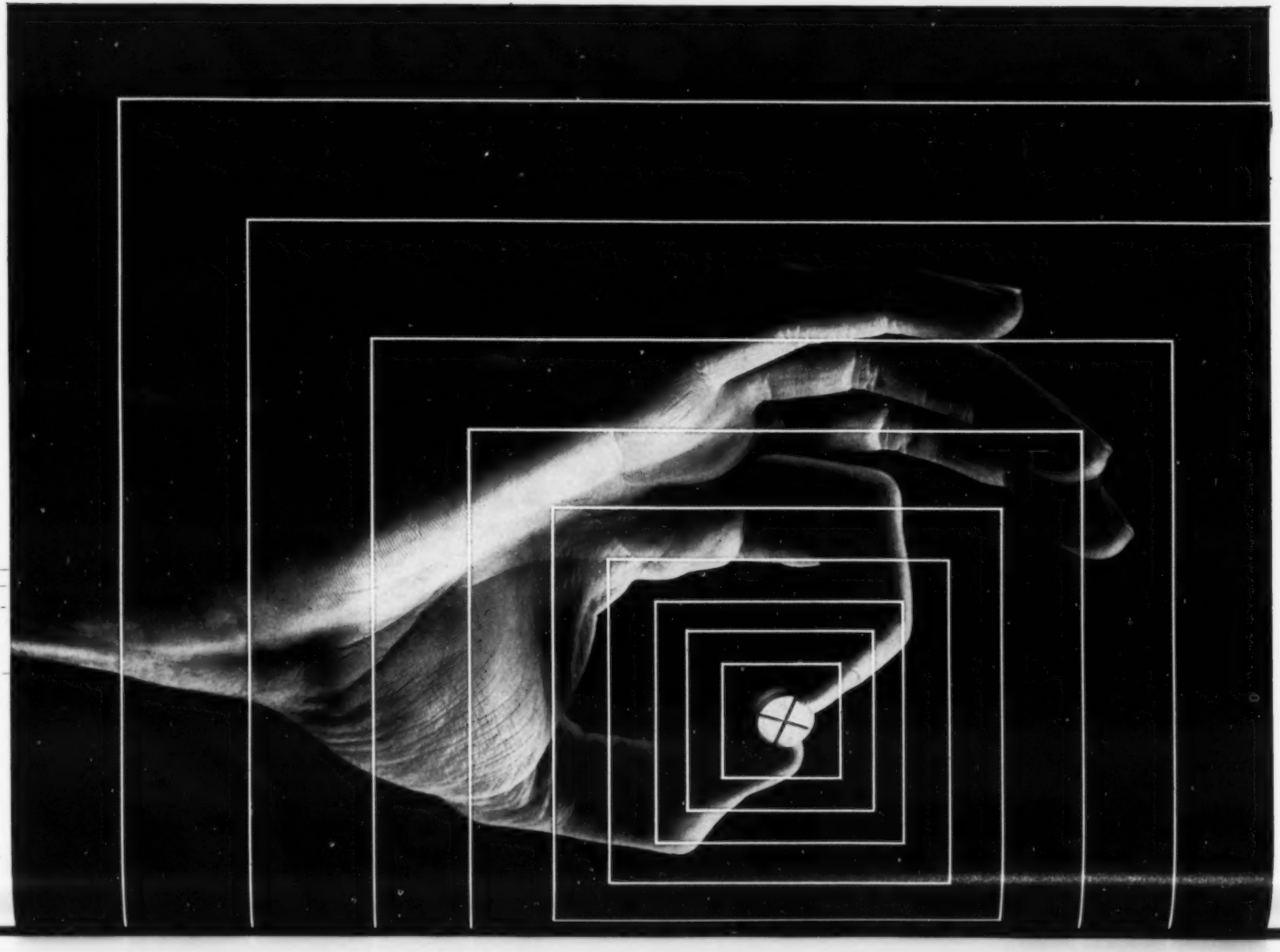
7. Catanzaro, F. JJ., Stetson, C. A., Morris, A. J., Chamovitz, R., Rammelkamp, C. H., Jr., Stolzer, B. L., and Perry, W. D.: The Role of the Streptococcus in the Pathogenesis of Rheumatic Fever. Am. J. Med. 27, 749-756, 1954.

8. Rammelkamp, Charles H., Jr.: The Natural History of Streptococcal Infections. Bull. N. Y. Acad. Med. 31: 103-112, 1955.

9. Griffith, George C.: The Epidemiology of Rheumatic Fever: Its Public Health Aspects. Am. J. Public Health 38: 682-688, 1948.

10. Schottstaedt, William W., Krause, James H., Foerster, David W., Dooley, Robert T., and Kelly, Florene C.: Host Factors Affecting Growth of B-Hemolytic Streptococcic in the Human Pharynx—Am. J. Med. Sci. 235: 23-32, 1958.

(600 N. E. 13, Oklahoma City, Oklahoma)



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SULFAMETHOXYPYRIDAZINE (3-SULFANILAMIDO-6-METHOXYPYRIDAZINE) LEDERLE

New authoritative studies prove that KYNEX dosage can be reduced even further than that recommended earlier.[1] Now, clinical evidence has established that a single (0.5 Gm.) tablet maintains therapeutic blood levels extending beyond 24 hours. Still more proof that KYNEX stands alone in sulfa performance—

- Lowest Oral Dose In Sulfa History—0.5 Gm. (1 tablet) daily in the usual patient for maintenance of therapeutic blood levels
- Higher Solubility—effective blood concentrations within an hour or two
- Effective Antibacterial Range—exceptional effectiveness in urinary tract infections
- Convenience—the low dose of 0.5 Gm. (1 tablet) per day offers optimum convenience and acceptance to patients

NEW DOSAGE. The recommended adult dose is 1 Gm. (2 tablets or 4 teaspoonfuls of syrup) the first day, followed by 0.5 Gm. (1 tablet or 2 teaspoonfuls of syrup) every day thereafter, or 1 Gm. every other day for mild to moderate infections. In severe infections where prompt, high blood levels are indicated, the initial dose should be 2 Gm. followed by 0.5 Gm. every 24 hours. Dosage in children, according to weight; i.e., a 40 lb. child should receive ¼ of the adult dosage. It is recommended that these dosages not be exceeded.

TABLETS: Each tablet contains 0.5 Gm. (7½ grains) of sulfamethoxypyridazine. Bottles of 24 and 100 tablets.

SYRUP: Each teaspoonful (5 cc.) of caramel-flavored syrup contains 250 mg. of sulfamethoxypyridazine. Bottle of 4 fl. oz.

1. Nichols, R. L. and Finland, M.: *J. Clin. Med.* 49:410, 1957.

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, NEW YORK

*Reg. U. S. Pat. Off.

25 YEARS AGO

Articles published in *The Journal* of the Oklahoma State Medical Association March, 1933.

A METHOD OF DETERMINING PERCENTAGE OF PARTIAL DISABILITY FOR THE OKLAHOMA STATE INDUSTRIAL COURT

Earl D. McBride, M.D., F.A.C.S.
Associate Professor Orthopedic Surgery
University of Oklahoma
Oklahoma City

"*The physician and the industrial court:* When an injured employee has sustained a disability of a permanent nature, the industrial courts expect the physician to determine the extent of disability. They depend on him to express in percentage the permanent or partial permanent loss, *i.e.*, he is to determine the functional ability less a disability.

"This involves the physician in a matter that is without the usual sphere of his medical training. The average physician is not experienced in industrial court activities and the problem of interpreting loss of function in mathematical or monetary values is entirely foreign to his customary reasoning of prognostic determination. Since he probably has never done hard manual labor it is difficult for him to determine the ability of an injured laborer to perform his duties. The average physician is not accustomed to being definite. In his practice he deals constantly with uncertainties. He is well acquainted with, and often makes indefinite patronizing expressions, optomistic and sympathetic for the psychological benefit of the patient. When confronted, therefore, with a problem which has on the one side a claimant who expects monetary remuneration for damages to his body, and on the other side by an employer who is forced by law to pay for such damages, the medical opinion immediately becomes valuable and all statements made must be such as to avoid prejudice and error.

"It behooves the physician to be strictly impartial. However, testimony and opinions are rendered in every industrial court which show a wide variation in mathematical evaluations of percentages of partial disability. This cannot be remedied unless some definite system of reasoning can be evolved. It is the writer's contention that disability should be based on functional loss and not upon anatomical and physiological imperfections. The disability should be evaluated upon what the man cannot do because of anatomical and physiological imperfections, not upon the extent of their existence. For instance, pain, atrophy or shortening of an extremity, measure the disability only to the extent that they interfere with such functions as quickness of action, coordination of movements, strength or security. Thus, we would have to consider two elements in reasoning a determination of evaluation. First, the contributing factors of anatomical and physiological damage. Second, the functional loss resulting from these imperfections.

"*Contributing Factors:* The contributing factors of a partial disability claim, *i.e.*, the anatomical and physiological deficiencies, for which an accident of industry is responsible are revealed by physical examination. For instance, a fractured bone that has healed in angulation necessarily disturbs mechanical perfection of the part. It may or may not interfere with use of the part in labor. If in the examiner's opinion it does interfere with function, the extent of this loss may be expressed in terms of percentage representing a limitation of the normal service of the part of the individual, and therefore, a diminished earning capacity. . . .

"*Functional Factors:* Functional factors pertaining to the ability of an individual to use his limbs and body in ordinary manual labor at normal wage earning capacity are as follows:

1. Quickness of action.
2. Coordination of movements.
3. Strength
4. Security.
5. Endurance.
6. Safety.
7. Ability to secure employment. . . .

"*Rating of Functional Factors:* It is reasonable that if each of these functional factors enter into the disability of the individual, then each should bear its relation to the evaluated loss. No single factor alone is responsible for total disability. For instance, if the disabled member of the body is 100 per cent weak, quickness of action, coordination of movements, security and endurance are also affected, but not necessarily to the amount of 100 per cent in respect to performing ordinary manual labor. It is necessary, therefore, that each of these factors should be given an evaluation according to its relative importance to the working capacity of the individual. The slowing up of activity and awkwardness are not important to the earning capacity as weakness in lifting power or the insecurity of holding, pulling or pushing power. The factors of diminished endurance and endangering safety are not to be rated quite as high as the damaging effect of lessening the individual's chance of securing future employment. The relative value of any of these factors will vary in many ways, in various cases. If each factor is given a percentage

value and the sum of all factors is 100, then even though relative importance may vary, the percentage of loss of the part as a whole will be equitably estimated. There may be a difference of opinion on the individual functional values, but if each is a portion of 100%, the resulting permanent loss of each will total a proportional percentage. . . .

Conclusions

"The writer has been stimulated to study out some definite ideals on this subject because it seems that dealings with industrial claims have come to be a part of the physician's duties and that the profession should take some steps to place it on just as honorable and scientific basis as any other part of medical practice.

It is hoped that the suggestions here developed will inspire others to continue the study. . . . "

Editorial Notes—Personal and General

" . . . KAY COUNTY MEDICAL SOCIETY met in January and elected the following officers for 1933: President, Dr. Merle C. Clift, Blackwell; Vice President, Dr. W. S. White, Blackwell; Secretary-Treasurer, Dr. L. H. Becker, Blackwell. . . .

"GRADY COUNTY MEDICAL SOCIETY elected the following officers at their January meeting to serve for 1933: President, Dr. J. F. Renegar, Tuttle; First Vice President, Dr. C. P. Mitchell, Chickasha; Second Vice President, Mr. C. P. Cox, Ninnekah; Secretary-Treasurer, Dr. L. E. Woods, Chickasha; Delegate, Dr. Roy E. Emanuel, Chickasha; Censor, Dr. D. S. Downey, Chickasha. . . ."

"DR. CURT von WEDEL, Oklahoma City, announces the establishment of a department of Internal Medicine and Diagnosis, under the supervision of Dr. Harry A. Daniels, formerly of Rochester, Minnesota. . . .

"JACKSON COUNTY MEDICAL SOCIETY elected the following officers to serve for 1933: Doctors, J. S. Stultz, Altus, president; C. G. Spears, Altus, vice president; E. W. Mabry, Altus, re-elected secretary; E. S. Crowe, Olustee, delegate; R. F. Brown, Altus, alternate. . . .

"COMANCHE COUNTY MEDICAL SOCIETY elected the following officers to serve for 1933: President, Dr. Thomas R. Lutner, Lawton; Vice President, Dr. L. W. Ferguson, Lawton; Secretary-Treasurer, Dr. E. Brent Mitchell, Lawton; Censors, Doctors J. W. Malcolm, H. A. Angus and P. G. Dunlap, all of Lawton. . . .

"BECKHAM COUNTY MEDICAL SOCIETY held a meeting at the Tisdal Hospital, Elk City, and reorganized their County Medical Society, in December. The following officers were elected for 1933: President, Dr. V. C. Tisdal, Elk City; Vice President, Dr. Phil DeVaney, Sayre; Secretary-Treasurer, Dr. C. F. Jones, Erick; Censors, Drs. H. K. Speed, Sayre; O. C. Standifer, Elk City, and R. C. McGregory, Erick. . . ."

"DRS. W. P. AND E. HALSELL FITE, Muskogee, attended the meeting of the American College of Surgeons held in St. Louis, in October."

Book Review

Healthful School Living. A report of the Joint Committee on Health Problems in Education of the National Education Association and the American Medical Association. Cloth binding, $5.00. Copyright, 1957.

Healthful School Living completes a trilogy of publications relating to the school health program. The other two are *Health Education* (1924) and *School Health Services* (1953). The three works compliment one another in supplying teachers, school administrators, physicians, dentists, nurses, and others concerned with the health of the school child, with a synthesis of medical and educational viewpoints.

This particular volume emphasizes solutions to health problems affecting pupils as they live and learn at school. Useful information is gathered here on such matters as school housekeeping, the prevention of accidents, water supply and waste disposal, heating, ventilating, and lighting. Chapters are included on the health aspects of the school lunch, school building construction, and physical education. There are significant suggestions for teachers and administrators on health problems involved in the organization of the school day, the health implications of various classroom procedures, and the special needs of rural schools.

Particular emphasis is placed on the administration in a school to assure healthful school living with space devoted to general administrative principles, school health policies, organization of the school health program, the school health budget, and health personnel and their responsibilities.

PHYSICIAN PLACEMENT

General Practice

Jack L. Coats, M.D., 1414-A East 17th Place, Tulsa, Oklahoma, age 29, married, veteran, University of Oklahoma School of medicine 1957, will be available July 1, 1958.

Sam Davis, M.D., 1605 N.E. 38, Oklahoma City, age 33, married, graduated from University of Oklahoma School of Medicine, 1956, veteran, will be available July 1, 1958.

General Surgery

Duane H. Dougherty, M.D., 201 Avon Road, Tonawanda, New York, age 30, married, veteran, New York University 1953, board qualified, will be available July, 1958.

Charles Edward Selah, M.D., Huey P. Long Charity Hospital, Pineville, Louisiana, age 31, married, will complete board requirements in June, 1958, veteran, graduated from Tulane 1951, will be available July 1, 1958.

Ralph L. Hopp, M.D., 338 E. Kingsley, Ann Arbor, Michigan. Married, veteran, Board Certificate held in General Surgery. Graduated Indiana University, 1950, will be available July 1, 1958.

Internal Medicine

Bartis M. Kent, M.D., 255 Koser, Iowa City, Iowa, age 32, married, Baylor, 1948. Three year residency at Baylor, veteran, available July, 1958.

Locum Tenens

Jack David Shirley, M.D., 430 Bellevue, Lafayette, Louisiana, age 27, married, will be inducted into U. S. Navy, October 6, 1958, graduated from University of Oklahoma in 1956, would like position for three months doing general practice. iWll be available July 1, 1958.

Neurosurgery

Bahif S. Salibi, M.D. (Currently Captain, MC U. S. Army) 121 Evac. Hosp., APO 971, San Francisco, California, age 35, married, Harvard, 1950, Board qualified in Neurosurgery, except for the two years of private practice required by the Boards, available October, 1958.

Obstetrics & Gynecology

Herbert Claiborne Jones, M.D., Jr., Box 166, University Hospital Charlottesville, Virginia, age 30, married, University of Virginia 1951, finishing board qualifications, will be available Spring, 1958.

Orthopedic Surgery

H. N. Hamilton, M.D., 13 Evergreen Road, Little Rock, Arkansas, age 36, married, veteran. Johns Hopkins, 1945, finishing Orthopedic Residency July 1958 and will be available at that time.

Thoracic and Cardiovascular Surgery

Wayne E. Hird, M.D., McGuire VA Hospital, Richmond 19, Virginia, age 31, married, Korean veteran, University of Kansas 1950, Board qualified in General Surgery, Thoracic and Cardiovascular exams will be completed by next July. Will be available July, 1958.

CLASSIFIED ADS

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WANT TO BUY your surplus equipment. We buy, sell,trade medical apparatus. Largest stock of good used medical devices in the Southwest. Expert repairs on X-ray and electromedical machines. Tell us about your equipment problems. TEX-RAY CO., opposite St. Paul's Hospital at 3305 Bryan Street, Dallas.

PHYSICIAN'S OFFICE EQUIPMENT: Medical Arts Building, Oklahoma City, available in nice office; two examining tables and stools; desk; reception room furniture; filing equipment; bookcase; refrigerator; scales; sterilizer; many small items; $500. Write Key F, c/o THE JOURNAL, P.O. Box 9696, Shartel Station, Oklahoma City, Oklahoma.

CLINIC LOANS: If you are planning to build a clinic, and need to secure financing, call Pat Allen, WI 2-2402 or write 1201 Classen Dr., Oklahoma City.

PHYSICIAN WANTED, State Veterans Hospital, Sulphur, approx. $900 per month plus other benefits. Contact C. E. Bates, M.D., Box 200, Sulphur, phone 851.

FOR SALE: Office equipment consisting of reception, consulting, examining, supply room. This office is located in northeast Oklahoma and the rent is fifty dollars per month if you should desire to use the present building. There is an opening here for another young doctor and this office could be ready for business immediately. The office equipment price is $2,000. If interested contact JOURNAL.

FOR SALE: Young Cystoscopic table, 100 M A rotating anode tube, controls, transformer, cossettes (complete unit) 3 years old—$2,500.00. Springer Clinic, Tulsa, Oklahoma, LU 7-6621.

WANTED PHYSICIAN: recent graduate as assistant in industrial surgery. Beginning July 1, 1958. Apply Glass-Nelson Clinic, Tulsa, Oklahoma, P. O. Box 3718.